FINAL HUMAN HEALTH RISK ASSESSMENT ADDENDUM
HORSESHOE ROAD COMPLEX SITE
SAYREVILLE, NEW JERSEY

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SUBJECT: Final Human Health Risk Assessment Addendum
Horseshoe Road Complex Site
Remedial Investigation/Feasibility Study
Sayreville, New Jersey

Dear Mr. Osolin:

CDM Federal Programs Corporation (CDM Federal), on behalf of our entire RAC II Team, is pleased to submit the attached Final Human Health Risk Assessment Addendum for the Horseshoe Road Complex Site as partial fulfillment of Subtask No. 7.1 of the Statement of Work.

If you have any questions regarding this submittal, please contact Joseph Mayo at (212) 785-9123 or myself at (908) 757-9500.

Very truly yours,
CDM FEDERAL PROGRAMS CORPORATION

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ACRONYMS AND ABBREVIATIONS

ADC	-	Atlantic Development Corporation
AOC	-	Area of Concern
ARARs	-	Applicable or Relevant and Appropriate Requirements
ARC	-	Atlantic Resource Corporation
ARCS	-	Alternative Remedial Contracting Strategy
AT	-	Averaging Time
BW	-	Body Weight
CDI	-	Chronic Daily Intake
CDM Federal	-	CDM Federal Programs Corporation
CERCLA	-	Comprehensive Environmental Response, Compensation, and Liability Act
COC	-	Chemical of Concern
COPC	-	Chemical of Potential Concern
CT	-	Central Tendency
EA	-	Ecological Assessment
ED	-	Exposure Duration
EF	-	Exposure Frequency
EPA	-	United States Environmental Protection Agency
ET	-	Exposure Time
FDA	-	Food and Drug Administration
FS	-	Feasibility Study
HEAST	-	Health Effects Assessment Summary Tables
HHRA	-	Human Health Risk Assessment
HRDD	-	Horseshoe Road Drum Dump
IR	-	Ingestion Rate; Inhalation Rate
IRIS	-	Integrated Risk Information System
LOAEL	-	Lowest-Observed-Adverse-Effect-Level
MCL	-	Maximum Contaminant Level
MCUA	-	Middlesex County Utilities Authority
NCEA	-	National Center for Environmental Assessment
NCP	-	National Oil and Hazardous Substance Pollution Contingency Plan (NCP)
NOAEL	-	No Observed Adverse Effect Level
NJDEP	-	New Jersey Department of Environmental Protection
NPL	-	National Priorities List
PAH	-	Polycyclic Aromatic Hydrocarbon
PAR	-	Pathway Analysis Report

PCB	-	Polychlorinated Biphenyl
PRG	-	Preliminary Remediation Goal
RA	-	Risk Assessment
RAGS	-	Risk Assessment Guidance for Superfund
RFC	-	Reference Concentration
RFD	-	Reference Dose
RI	-	Remedial Investigation
RME	-	Reasonable Maximum Exposure
RR	-	Raritan River
SF	-	Slope Factors
SPD	-	Sayreville Pesticide Dump
SVOC	-	Semi-volatile Organic Compound
TAL	-	Target Analyte List
TCL	-	Target Compound List
TCLP	-	Toxicity Characteristic Leaching Procedure
TDS	-	Total Dissolved Solids
UCL	-	Upper Confidence Limit
VOC	-	Volatile Organic Compound

1.0 INTRODUCTION

CDM FEDERAL PROGRAMS CORPORATION (CDM Federal) received Work Assignment Number 085-2COBT under the ARCS II program to perform a Remedial Investigation/Feasibility Study (RI/FS), including a Risk Assessment (RA) for the United States Environmental Protection Agency, Region II (EPA) at the Horseshoe Road Complex Superfund site located in Sayreville, New Jersey. This assignment was rolled over to work assignment number 013-RICO-O2BT under the RACII program to finalize the RI/FS and Risk Assessment. The purpose of the RI/FS is to evaluate the overall nature and extent of contamination at the site and to develop and evaluate remedial alternatives, as appropriate. The purpose of the RA is to provide an analysis of baseline risks to determine the need for remedial action at the site and to serve as a basis for determining cleanup levels which will adequately protect human health and the environment.

A baseline Human Health Risk Assessment (HHRA) and an Ecological Risk Assessment (ERA) have been completed. This document is an addendum to the baseline HHRA, and considers additional shellfish data collected from the Raritan River after the completion of the baseline HHRA. These shellfish data were collected to provide site-specific tissue concentrations to be used in the risk assessment in place of modeled data.

1.1 SCOPE OF RISK ASSESSMENT

Task 5.5.2 of the Final Work Plan (dated June 1997) required the preparation and submittal to EPA of a Human Health Risk Assessment (HHRA) which was completed October 1999 (CDM Federal, 1999a). CDM submitted a Pathway Analysis Report (PAR) to EPA in July 1998. The PAR specified the conceptual approach that would be used to evaluate the potential human health risks associated with the site. The following are the components of the HHRA as specified in the work plan:

- Data Collection and Evaluation
- Exposure Assessment
- Toxicity Assessment
- Risk Characterization
- Uncertainties in Risk Assessment

This addendum to the baseline HHRA follows the same conceptual approach.

DATA COLLECTION AND EVALUATION

The first step of the risk assessment, Data Collection and Evaluation, is presented in Section 2.0 of this report. This section includes a summary of shellfish data collected after the completion of the baseline HHRA (Appendix B). A subset of the chemicals of concern (COCs) identified in the muscle tissue of shellfish samples from the Raritan River (AOC6) were selected for detailed analysis. The primary selection criteria for these chemicals included 1) the chemical concentrations; 2) a chemical concentration-toxicity screen (Appendix C); 3) the frequencies of detection; 4) the physical/chemical parameters; 5) the degree of toxicity, mobility, and persistence in the environment; and 6) historical information about site activities and the chemicals reliably associated with these activities. The COCs are presented in Appendix D.

EXPOSURE ASSESSMENT

In the second step, Exposure Assessment, qualitative or quantitative estimates of the magnitude, frequency, duration, and routes of exposure were made. The pathways through which chemical contaminants migrate from potential sources to existing receptors were identified. Receptor groups (i.e., human populations) that might potentially be exposed as a result of the presence of one or more chemicals in the environment were also identified.

Exposure point concentrations for COCs are typically estimated based on the 95 percent Upper Confidence Limit (UCL) on the arithmetic mean. However, for this addendum HHRA, the 95 percent UCL was not used to calculate the exposure point concentrations because fewer than ten samples were collected. Data sets with fewer than ten samples provide poor estimates of the mean concentration because there may be a significant difference between the sample mean and the 95 percent UCL (EPA, 1992a), resulting in a 95 percent UCL greater than the maximum concentration. Therefore, the maximum detected concentrations were used to prevent overestimation of potential human health impacts.

Daily chemical intakes via the exposure route were quantitatively evaluated based on the maximum concentration and the site-specific, medium-specific, and receptor-specific intake variables. As previously stated, exposures were estimated for the reasonable maximum case exposure scenario (RME), which in this case employs the maximum concentration and RME assumptions. It should be noted that the risk assessment assumes that no reduction in exposure concentrations occurs due to natural physical/chemical processes, site

remediation or institutional controls. The results of this evaluation are provided in the Exposure Assessment (Section 3.0) of the risk assessment.

TOXICITY ASSESSMENT

The third step of the risk assessment is the Toxicity Assessment. The purpose of the toxicity assessment was to weigh available toxicological evidence regarding the potential for a particular chemical contaminant to cause adverse health effects in exposed individuals and to provide, where possible, an estimate of the relationship between the extent of exposure to a chemical contaminant and the increased likelihood and/or severity of adverse health effects (EPA, 1989a).

EPA has performed the toxicity assessment step for numerous chemicals and has made available the resulting toxicity information and toxicity values, which have undergone extensive peer review; however, data analysis and interpretation are still required. These established toxicity values were obtained from the Integrated Risk Information System (IRIS) data base (August 2000), which is updated monthly, or from the Health Effects Assessment Summary Tables (HEAST) FY 1997 - Annual, if no value was found in IRIS. The Superfund National Center for Environmental Assessment (NCEA) was consulted for other specific chemical toxicity values, as directed by HEAST, when no value was shown.

A toxicity profile for each COC was developed using EPA toxicity assessments and accompanying values. These profiles were presented in the baseline HHRA dated October 1999 (CDM Federal, 1999a). Additional toxicity profiles that were not included in the baseline HHRA are included in Appendix E of this addendum HHRA. The toxicity values and the limitations of use of the toxicity values have been described in the Toxicity Assessment (Section 4.0) of the risk assessment. Chemicals without toxicity data are qualitatively discussed in Section 5.0, Risk Characterization.

RISK CHARACTERIZATION

In the last step of the risk assessment process, Risk Characterization, the chronic daily intake for each chemical to which the receptor group might be exposed was compared with concentrations known or suspected to present some health risk or hazard. Quantitative estimates of the carcinogenic risks and noncarcinogenic health effects associated with each exposure pathway are presented for current and potential future land uses of the site.

The risks resulting from exposures to carcinogens were estimated based on the following assumptions (EPA, 1989):

- A linear relationship exists between the intake of a carcinogenic substance over a lifetime and the risk of cancer (the linearized multistage model of carcinogenesis assumes that the dose-response relationship will be linear in the low-dose portion of the multistage model dose-response curve).

- Cancer risks from exposures to all carcinogens via all intake routes are additive.

The potential for noncarcinogenic effects was evaluated by comparing an exposure level over a specified time period with a reference dose derived for a similar exposure period.

Section 5.0 of this risk assessment presents the risk characterization, and a comparison of the risk characterization results to the baseline HHRA results. Spreadsheet calculations containing complete results are presented in Standard Tables 7 and 8 of this report.

UNCERTAINTIES IN RISK ASSESSMENT

Because of the number of assumptions required during the risk assessment process, some degree of uncertainty is inevitably associated with the risk and hazard estimates. Additionally, because shellfish are migratory in nature, the contaminants present in the crab tissue may have been derived from other areas. In the future, NJDEP's Toxics in Biota Monitoring Program will have crab tissue data available for identifying background levels in shellfish in the Raritan River. These data will enable us to determine whether the contamination in the shellfish at this site is comparable to other areas of the river.

These uncertainties have been addressed qualitatively in Section 6.0, Uncertainties in Risk Assessment.

SUMMARY

A summary of the results of the risk assessment is presented in Section 7.0 of this report.

REFERENCES

This addendum HHRA was prepared in accordance with EPA Region II and other EPA risk assessment guidance documents and the on-line data base listed below.

- *Risk Assessment Guidance for Superfund: Human Health Evaluation Manual, Part A* (EPA, 1989a).

- *Risk Assessment Guidance for Superfund: Human Health Evaluation Manual, Part D* (EPA, 1998a).

- *Exposure Factors Handbook* (EPA, 1996).

- *Integrated Risk Information System (On-line data base of toxicity measures)* (EPA, 2000).

1.2 SITE DESCRIPTION AND HISTORY

The Horseshoe Road Complex Site is located in Sayreville (Lots 1.01 and 1.03 in Block 246 and Lots 2.02 through 2.04 in Block 256), Middlesex County, New Jersey (Figure 1). The abandoned site (Figure 2), situated near the Raritan River, includes three adjoining areas of concern: (1) the Horseshoe Road Drum Dump (HRDD); (2) the Atlantic Development Corporation (ADC) Area; and (3) the Sayreville Pesticide Dump (SPD). The Atlantic Resource Corporation (ARC) is also located in the complex, but it is not part of the National Priorities List (NPL) site. The site, which consists of several abandoned industrial buildings and warehouses, is bordered to the north by the Raritan River, to the east by Conrail railroad tracks and easement, and to the west and south by wooded areas.

The area surrounding the site is used for both residential and industrial purposes. At least 47 residences are located within a one-mile radius of the site, while several hundred single family and multi-resident buildings are located within a two-mile radius. New Jersey Steel Corporation operates a facility approximately one-half mile to the southwest. The Middlesex County Utilities Authority (MCUA) operates a water treatment plant on the northern side of the site and a MCUA trunk line and a maintenance right of way cuts through the ARC and ADC properties. The Sayreville Water Company, which supplies water to approximately 14,000 people, maintains wells, recharge lagoons, and force mains several miles south of the site on Bordentown Road.

For over 30 years, various operations were conducted at the Horseshoe Road Complex including the manufacturing of epoxy resins, roofing materials, paint pigments, and pharmaceuticals. Poor waste handling practices and the dumping of waste materials resulted in site-wide contamination. In addition, releases of copper, lead, methoxychlor, lindane, phenol, bis(2-ethylhexyl)phthalate, chloroform, 1,2-dichloroethane, and mercury to the Raritan River have also been reported.

Investigations by EPA and the New Jersey Department of Environmental Protection (NJDEP) have documented contamination of the site's surface and subsurface soil, surface water and sediment, and groundwater. Elevated levels of volatile organic, semivolatile organic, pesticide, dioxin, polychlorinated biphenyls (PCBs), and inorganic contamination have been detected in the site media.

To date, EPA has conducted more than nine removal actions that have addressed immediate public health threats and that have restricted site access. Removal actions, which began in 1987, included the removal of 3,000 drums, both buried and located on the ground surface, the remediation of mercury and dioxin spills, the removal and disposal of tank and vat materials, and the excavation and disposal of contaminated soils and debris.

Under EPA authorization, CDM Federal initiated field investigation activities in October of 1997, with completion in August of 1998. A detailed description of the investigation is presented in the Final Remedial Investigation Report, completed May 1999 (CDM Federal, 1999b). Additional sampling was conducted in the Fall of 1999 through the Spring of 2000 to provide specific data for the Ecological Assessment (EA). The results of this sampling are presented in the Data Summary Report dated July, 2000 (CDM Federal, 2000).

The site was proposed for inclusion on the EPA Superfund NPL in June 1993 and was listed in September 1995.

2.0 DATA COLLECTION AND EVALUATION

In the first step of the HHRA, Hazard Identification, the samples collected and the chemicals analyzed are discussed. The addendum HHRA includes a summary of the locations sampled, the number of samples collected, and the analyses conducted on the samples. For the addendum RI, 12 samples of shellfish muscle were collected from 11 sites, including 2 background sites. Sample locations are presented in Figure 3 and background sample locations are presented in Figure 4. These shellfish samples were collected to provide site-specific tissue concentrations for use in the risk assessment in place of modeled data.

2.1 MEDIA TO BE EVALUATED

The environmental media to be quantitatively evaluated in the addendum HHRA is shellfish in surface water. Twelve muscle tissue and hepatopancreas samples from shellfish were collected from 11 locations in September, 1999. All 11 of these locations were found in the Raritan River (AOC6). Each sample was analyzed for TCL VOCs, TCL Extractables, and TAL Inorganics. Muscle tissue samples will be used in this addendum risk assessment since this is the portion of the shellfish that humans consume. The hepatopancreas samples were collected for environmental risk evaluation. The shellfish muscle data are presented in Appendix B.

Included in the 12 samples is a composite sample (RCMCOMP1) that consists of shellfish from sites RCM04, RCM05, and RCM07. The composite sample was not used in this risk assessment because these three sample sites are already represented by individual samples from each of the three locations. Data from the composite sample are presented in Appendix B.

Samples RCM01 and RCM19 were collected as background samples. These background samples were not used to screen out constituents of concern because they were collected from a tidal area and are presented for comparison purposes only (Standard Table 2). In the future, NJDEP's Toxics in Biota Monitoring Program will have crab tissue data available for identifying background levels in shellfish from the Raritan River.

2.2 TREATMENT OF DATA

The summary of the data presented in Standard Table 2 includes the frequency of detection, the range of detected concentrations, the location of the maximum detected concentration, and the range of non-detect concentrations for each detected chemical. The

frequency of detection is reported as the number of samples with detected concentrations divided by the number of analyzed samples.

Blanks, including field, trip, and laboratory, and rejected data (i.e., qualified with "R") were not included in the frequency tally or range of concentrations.

2.2.1 DATA QUALITY

As part of the data evaluation process, the quality of data was evaluated in the data validation phase. All RI data were validated in accordance with EPA Region II data validation protocols. However, it should be noted that the data from certain samples and analytes were qualified. In general, data with qualifiers that indicate uncertainties in concentrations but not identity will be utilized in this risk assessment. Rejected data, qualified with an "R", will not used in this risk assessment because the chemical's identity and concentration are uncertain. Data qualified with a "U" will be used in this risk assessment, as appropriate, in producing data summary tables.

The data qualifiers associated with the site's database are as follows:

- The "*" qualifier indicates for inorganics that duplicate analysis was not within control limits.
- The "J" qualifier indicates for all chemicals that the reported concentration is estimated.
- The "B" qualifier indicates for organics that the reported concentration is estimated because it was detected in both the sample and in the associated blank; for inorganics, the "B" qualifier indicates that the reported value is less than the contract required detection limit but greater than the instrument detection limit.
- The "E" qualifier indicates for organics that the concentration exceeds the calibration range of the gas chromatograph/mass spectrometry (GC/MS) instrument; for inorganics, the "E" qualifier indicates that the value is estimated due to matrix interferences.
- The "N" qualifier for organics indicates that there is only presumptive evidence for their presence; for inorganics, the "N" qualifier indicates that the spiked sample recovery is not within control limits.

- The "U" qualifier for all chemicals indicates that the chemical was not detected at the reported detection limit.

- The "M" qualifier for inorganics indicates that duplicate injection precision was not met.

- The "P" qualifier for organics indicates that the difference for the detected concentration of a pesticide/ Aroclor target analyte is greater than 25% between the two GC columns.

2.3 CRITERIA FOR THE SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Because of the number of chemicals detected at the site, those retained for quantitative analysis in the HHRA addendum were selected as the most significant (i.e., greatest contributors to risks/hazards). The primary selection criteria for these chemicals included 1) the chemical concentrations; 2) a chemical concentration-toxicity screen; 3) the frequencies of detection; 4) the physical/chemical parameters; 5) the degree of toxicity, mobility, and persistence in the environment; and 6) historical information about site activities and the chemicals reliably associated with these activities.

The potential health impact of a chemical is influenced by the relationship of concentration and toxicity. A chemical detected at high concentrations that may exhibit low noncarcinogenic toxicity may have less impact on human health than a potential carcinogen detected at relatively low concentrations. Therefore, a chemical concentration - toxicity screening procedure was performed for all chemicals detected to aid in the determination of which chemicals were likely to contribute significantly to potential risks and hazards (Appendix C).

Individual chemical scores (or risk factors) were calculated as follows:

$$R_{ij} = (C_{ij})\,(T_{ij})$$

Where:

R_{ij} = risk factor for chemical i in medium j
C_{ij} = concentration of chemical i in medium j
T_{ij} = toxicity value for chemical i in medium j
(i.e., slope factor or 1/oral reference dose)

For conservatism, the maximum detected concentration of each chemical was used in the calculation (EPA, 1989). The chemical-specific risk factors per area were summed to obtain a total risk factor for all chemicals for each area. Separate total risk factors were calculated for carcinogens (using the appropriate slope factors) and noncarcinogens (using the appropriate oral reference doses). The ratio of the risk factor for each chemical to the total risk factor provided the relative contribution from each chemical. A contribution of 1 percent was used as a lower limit and chemicals contributing at least 1 percent were selected as COCs (EPA, 1989). Additionally, chemicals detected in shellfish muscle were screened against Region III RBGs (fish) to insure that all chemicals were included, as appropriate.

For the evaluation of chromium in the concentration-toxicity screens, total chromium was speciated into its +3 and +6 valence states using a ratio of 6:1, respectively, per the IRIS data base.

The selected chemicals of concern (COCs) are presented in Appendix D.

3.0 EXPOSURE ASSESSMENT

The objective of this section is to present the analysis for selecting potential exposure pathways to be evaluated in the addendum HHRA. An exposure pathway analysis describes the transport of a chemical from the source of release to the exposed individual. An exposure pathway links the sources, locations, and types of environmental patterns to determine significant pathways of human exposure. As defined in EPA's Risk Assessment Guidance for Superfund (RAGS), an exposure pathway has four elements:

- Source and mechanism of chemical release
- Release or transport mechanism
- Point of potential human contact (exposure point)
- Exposure route at the contact point

For the baseline HHRA (CDM Federal, 1999a) the identification of potential release mechanisms and receiving media were determined utilizing site histories and data from existing reports. The fate and transport of the chemicals from release media were also considered to identify media that are receiving or that may receive site-related chemicals. Points of potential contact with chemically contaminated media (or sources) by human receptors were then considered.

For the addendum HHRA, the ingestion pathway was identified as the pathway through which chemical contaminants can migrate from shellfish to the existing receptors. Receptor groups (i.e., human populations) that may potentially be exposed as a result of the presence of one or more chemicals in the environment were identified. For the addendum HHRA, the adult resident was selected as the receptor who may be exposed via ingestion of the contaminated medium.

3.1 IDENTIFICATION OF EXPOSURE PATHWAYS

The exposure pathway for the addendum HHRA of the Horseshoe Road Complex Site is presented in Standard Table 1. Standard Table 4 presents the exposure variables to be used in the daily intake calculations for each complete exposure pathway.

Historical sampling at the site indicated surface water and sediment contamination. Onsite surface water and sediment (e.g., pond, stream, drainage channels and wetlands) and associated surface water run-off may currently be contacted by area residents/trespassers. Run-off from the site into the Raritan River may potentially pose a threat to residents ingesting shellfish caught in the river.

3.2 EXPOSURE POINT CONCENTRATIONS

Concentrations at potential exposure points (any point of potential contact with a contaminated medium) were developed for each COC in shellfish for use in calculation of daily intakes. Because of the uncertainty associated with any estimate of exposure concentration, the 95 percent UCL on the arithmetic mean is typically used for this variable. However, for this addendum HHRA, the 95 percent UCL was not used to calculate the exposure point concentrations because fewer than ten samples were collected. Data sets with fewer than ten samples provide poor estimates of the mean concentration because there may be a significant difference between the sample mean and the 95 percent UCL (EPA, 1992a), resulting in a 95 percent UCL greater than the maximum concentration. Therefore, the maximum detected concentrations were used to prevent overestimation of potential human health impacts.

Standard Table 3 presents the medium-specific exposure point concentration summaries.

3.3 CALCULATION OF DAILY INTAKES

To assess the potential carcinogenic risks and health hazards to human populations quantitatively based on the present-use and potential future-use scenarios discussed in Section 3.1, daily intakes were calculated. For daily intakes, intakes are averaged over a lifetime for carcinogenic chemicals and over the period of exposure for noncarcinogens. The daily intake is expressed in terms of the mass of the chemical contaminant per unit of body weight over the averaging time (mg chemical/kg body weight-day).

Equations presented and described in RAGS (EPA, 1989) were used to estimate daily intakes from ingestion exposures. These equations and values used for daily intake calculations are presented in Standard Table 4.

4.0 TOXICITY ASSESSMENT

In the toxicity assessment portion of the risk assessment, the relationship between the potential level of exposure (dose) and the likelihood and/or severity of adverse effects (response) will be evaluated. As part of this evaluation, available toxicity values or dose/response parameters for the chemicals detected at the site will be compiled. These dose/response parameters will be used in the chemical concentration-toxicity screens and integrated with chemical intake levels derived in exposure assessment to characterize the level of potential risks and health effects.

Dose/response parameters have been developed by EPA for the evaluation of both noncarcinogenic and carcinogenic effects of exposure to humans. The oral reference doses (RfDs) are the toxicity values used to evaluate noncarcinogenic effects resulting from exposure. The oral cancer slope factors (CSFs) are used to evaluate potential carcinogenic effects. Oral RfDs, as well as SFs derived for oral exposures, are available through EPA's on-line Integrated Risk Information System (IRIS) and Health Effects Assessment Summary Tables (HEAST) Annual FY-1997. When a value was not available through these sources, the EPA's National Center for Environmental Assessment (NCEA) was consulted.

4.1 NONCARCINOGENIC EFFECTS

4.1.1 DEFINITION AND DERIVATION OF REFERENCE DOSES

Toxicity values are available depending on the exposure route (oral or inhalation), the critical effect, and the length of exposure (e.g., chronic) to be evaluated. Chronic and subchronic oral and inhalation RfDs may be used to evaluate noncarcinogenic effects. A chronic RfD is defined as an estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of harmful effects during a lifetime. Chronic RfDs are specifically developed to be protective of long-term exposure to a chemical, and are defined as exposure periods exceeding seven years (approximately ten percent of a human lifetime of 70 years). Subchronic RfDs are used to characterize potential noncarcinogenic effects associated with shorter-term exposure periods between 2 weeks and approximately 7 years.

RfDs are derived by EPA based on the concept of a threshold. For many noncarcinogenic effects, protective mechanisms may exist which must be overcome before an adverse effect is manifested. A range of exposure levels may be tolerated by an organism before an adverse effect occurs. In the development of the RfDs, human epidemiological and clinical

studies, and experimental animal studies are reviewed to identify the upper-bound of the tolerance range (i.e., maximum subthreshold level) which is protective of sensitive individuals in the population. The no observed adverse effect level (NOAEL) or lowest observed adverse effect level (LOAEL) is generally used to describe this level and is the basis for the derivation of the RfD. Uncertainty and modifying factors are then applied to the NOAEL, depending on the quality and the applicability of the available animal or human toxicity study, as the final step in the derivation of the RfD. The resultant oral RfD is expressed in terms of unit concentration of a chemical (mg) per unit body weight (kg) per unit time (day) or mg/kg/day.

4.1.2 RfDS FOR DETECTED CHEMICAL CONTAMINANTS

Chronic oral RfDs, primary target organs, and the uncertainty factors associated with them for chemicals detected in historical site investigations are presented in Standard Table 5. These RfDs were used in the concentration-toxicity screens to select contaminants of concern (COCs), and in the calculation of ingestion noncarcinogenic hazard quotients (Standard Table 7). No COCs were evaluated for inhalation exposures, therefore, no inhalation reference concentrations were applicable. In addition, no special case chemicals were evaluated, therefore, no toxicity values were applicable for special case chemicals.

4.2 CARCINOGENIC EFFECTS

4.2.1 DEFINITION AND DERIVATION OF SLOPE FACTORS

The carcinogenic slope factor and the accompanying weight-of-evidence classification are used to evaluate potential human carcinogenic risks associated with exposures. The hypothesized mechanism of carcinogenesis is based on the concept of nonthreshold effects (i.e., there is essentially no level of exposure to a chemical that does not pose some probability of generating a carcinogenic response).

In defining the potential carcinogenicity of a chemical contaminant to humans, EPA CERCLA first evaluates the sufficiency of evidence of carcinogenicity from available data. The evidence is characterized separately for human and animal studies as sufficient, limited, adequate, no data, or evidence of no effect. The characterizations of these two sets of data are evaluated in combination and the chemical is assigned a "weight-of-evidence" classification. EPA has five groups of classification which are as follows:

A	-	Human Carcinogen.
B1	-	Probable Human Carcinogen. Limited human data are available.
B2	-	Probable Human Carcinogen. Sufficient evidence of carcinogenicity

in animals and inadequate or no evidence in humans.

C - Possible Human Carcinogen.

D - Not Classifiable as to human carcinogenicity.

E - Evidence of noncarcinogenicity for humans.

For Group A, B1, and B2 carcinogens, EPA typically derives a carcinogenic slope factor. Slope factors for Class C carcinogens are derived on a case-by-case basis. The slope factor defines quantitatively the relationship between dose and response as the plausible upper-bound estimate of the probability of a response (i.e., development of cancer) per unit intake of a potential carcinogen over a lifetime.

The slope factor is derived by EPA by selecting the most appropriate data set, extrapolating to lower doses, determining equivalent human doses for the appropriate route of exposure (ingestion), and application of uncertainty factors. The resultant slope factor is expressed in terms of risk per unit concentration of the chemical (mg) per unit body weight (kg) per unit time (day) or $(mg/kg/day)^{-1}$.

4.2.2 SLOPE FACTORS FOR DETECTED CHEMICAL CONTAMINANTS

Oral slope factors and weight-of-evidence classifications for potentially carcinogenic chemicals detected in historical site investigations are presented in Standard Table 6. These cancer slope factors (CSFs) will be used in the concentration-toxicity screens to select contaminants of concern (COCs), and in the calculation of ingestion carcinogenic risks (Standard Table 8). No COCs were evaluated for inhalation exposures, therefore, no inhalation slope factors were applicable. In addition, no special case chemicals were evaluated, therefore, no toxicity values were applicable for special case chemicals.

5.0 RISK CHARACTERIZATION

In this section of the risk assessment, toxicity and exposure assessments will be integrated into quantitative and qualitative expressions of carcinogenic risk and noncarcinogenic hazards. The estimate of risk and hazard will be expressed numerically in spreadsheets contained in the Standard Tables 7, 8, and 9 in Appendix A.

The potential for noncarcinogenic effects was evaluated by comparing an exposure level over a specified time period with a reference dose derived for a similar exposure period. This ratio of exposure to toxicity is referred to as a hazard quotient. The hazard index is the sum of the HQs. This hazard index assumes that there is a level of exposure below which it is unlikely even for sensitive populations to experience adverse health effects. If the hazard index exceeds 1, there may be concern for potential noncancer effects, however, this value should not be interpreted as a probability. Generally, the greater the hazard index above unity, the greater the level of concern. Calculation of non-cancer hazards are presented in Standard Table 7.

Carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen. Per RAGS guidance, the slope factor converts estimated daily intakes averaged over a lifetime of exposure directly to incremental risk of an individual developing cancer. This carcinogenic risk estimate is generally an upper-bound value since the slope factor is often an upper 95th percentile confidence limit of the probability of response based on experimental animal data used in the multistage model. Calculation of cancer risks are presented in Standard Table 8.

In general, EPA recommends a target value or a risk range (i.e., hazard index = 1 or risk = 10^{-4} to 10^{-6}) as threshold values for potential human health impacts. The results presented in the spreadsheet calculations were compared to these target values. These values aid in determining the objectives of the baseline risk assessment which include determining whether additional response action is necessary at the site, by providing a basis for determining residual chemical levels that are adequately protective of human health, by providing a basis for comparing potential health impacts of various remedial alternatives, and to help support selection of the no-action remedial alternative, where appropriate.

Carcinogenic risks and noncarcinogenic hazard indices are summarized for the adult resident receptor from the ingestion of shellfish from the Raritan River (Standard Table 9). Standard Table 9 also includes surface water and sediment risk and hazard index results from the baseline HHRA.

5.1 QUANTITATIVE RESULTS OF CARCINOGENIC RISK AND NONCARCINOGENIC EFFECTS EVALUATION

The results of carcinogenic risk and noncarcinogenic hazard index calculations for current and future adult residents are presented in Standard Table 9. In this addendum HHRA, exposures to adult residents were evaluated for shellfish. In the baseline HHRA, exposures to adult residents were evaluated for surface water and sediment. The total risk and hazard index from the ingestion of shellfish are 5.9E-05 and 0.55, respectively. The total risk across all media and all exposure routes is 2.5E-04, primarily attributed to arsenic in sediment. The total hazard index across all media and all exposure routes is 1.8. The total HI for skin is 1.5, attributed to arsenic in sediment. See the baseline HHRA for the COPCs, media, and exposure points that trigger the need for cleanup.

This HHRA addendum was performed to replace modeled data used in the baseline risk assessment. These new data result in a greater number of COCs, with higher carcinogenic risks and noncarcinogenic hazard quotients for comparable constituents. A comparison of the baseline versus the addendum HHRA results are shown in Table 1.

Table 1: Comparison of Baseline to Addendum HHRA Risk Values for Shellfish Ingestion

	Hazard Quotient		Cancer Risk	
	Baseline	**Addendum**	**Baseline**	**Addendum**
Aluminum	--	--	--	--
Antimony	1.3E-09	3.1E-02	--	--
Arsenic	2.6E-07	3.0E-01	4.1E-10	4.6E-05
Copper	1.1E-07	--	--	--
Cadmium	--	7.1E-03	--	--
Manganese	2.2E-07	--	--	--
Selenium	--	2.3E-02	--	--
Silver	--	9.8E-03	--	--
Thallium	2.2E-07	--	--	--
Vanadium	--	--	--	--
Zinc	--	1.5E-02	--	--
4,4'-DDD	--	--	--	8.1E-07
4,4'-DDE	--	--	--	1.2E-06
bis(2-Ethylhexyl) Phthalate	--	1.4E-03	--	1.4E-07
Dieldrin	--	1.7E-02	--	4.7E-06
Heptachlor Epoxide	--	1.4E-01	--	5.8E-06
Total	***8.1E-07***	***5.5E-01***	***4.1E-10***	***5.9E-05***

5.2 QUALITATIVE ASSESSMENT OF CHEMICALS WITHOUT TOXICITY VALUES

The quantitative risk assessment of receptors who ingest shellfish does not include several compounds detected in the sampling event. Some compounds are essential nutrients (i.e., calcium, magnesium, potassium, and sodium) and others lacked sufficient toxicological data.

The inorganic compounds copper, lead, mercury and dimethyl phthalate could not be quantitatively evaluated due to a lack of USEPA toxicity factors. After IRIS was checked, the National Center for Environmental Assessment (NCEA) was contacted for toxicity information. No toxicity factors were available to quantitatively evaluate the oral route for these four chemicals.

Copper: This chemical is an essential element widely distributed in nature. Acute poisoning from ingestion of excessive amounts of oral copper salts may produce death. Symptoms include vomiting, hematemesis, hypotention, melena, coma, and jaundice. This chemical has been given a Group D weight-of-evidence classification. A toxicological profile for this chemical is located in Appendix E of the Final Horseshoe Road HHRA dated October 1999.

Lead: This chemical has been given a Group B2 weight-of-evidence classification. A toxicological profile for this chemical is located in Appendix E of the Final Horseshoe Road HHRA dated October 1999. A comparison of lead shellfish concentration in muscle tissue to FDA levels of concern is presented in Section 5.3.

Mercury: This chemical has been given a Group D weight-of-evidence classification. An inhalation RfC is available but not an oral RfD or cancer slope factor. A toxicological profile is located in Appendix E of this HHRA addendum. A comparison of mercury shellfish concentration in muscle tissue to FDA levels of concern is presented in Section 5.3.

Dimethyl phthalate: This chemical has been given a Group D weight-of-evidence classification. A toxicological profile for this chemical is located in Appendix E of this HHRA addendum.

The inability to quantitatively evaluate these chemicals is a source of uncertainty in the risk assessment because of the potential to underestimate risks and health impacts.

5.3 QUALITATIVE DISCUSSION OF APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS

Concentrations of the COCs were qualitatively compared to the Applicable or Relevant and Appropriate Requirements (ARARs). For the addendum HHRA, estimated daily intakes of COCs were compared to the Guidance Document for Trace Elements in Seafood, by the Food and Drug Administration (FDA, 1993). This document assists local and state health officials to determine the possible need to issue advisories or close water for fishing because of excessive metal contamination from arsenic, cadmium, chromium III, lead and nickel.

For arsenic, the document suggests a maximum tolerable daily intake of 130 µg/person/day. For individuals who chronically consume an average of 17 µg/day of shellfish, with an arsenic intake maximum of 130 µg/person/day, the arsenic level of concern would be 7.6 ppm. The maximum arsenic level detected in crab muscle tissue in the Raritan River was 1 ppm which is lower than the arsenic level of concern of 7.6 ppm. Using the maximum concentration of arsenic of 1 ppm, the arsenic intake is 17 µg/person/day which is significantly less than the maximum tolerable daily intake of 130 µg/person/day.

For cadmium, the document suggests a maximum tolerable daily intake of 55 µg/person/day. For individuals who chronically consume an average of 17 µg/day of shellfish, with a cadmium intake maximum of 55 µg/person/day, the cadmium level of concern would be 3.2 ppm. The maximum cadmium level detected in crab muscle tissue in the Raritan River was 0.08 ppm, which is lower than the cadmium level of concern of 3.2 ppm. At the maximum cadmium level detected in crab muscle tissue in the Raritan River, the corresponding cadmium intake is 1.4 µg/person/day. This value is lower than the maximum tolerable daily intake of 55 µg/person/day.

For chromium, the document suggests a maximum tolerable daily intake of 200 µg/person/day. For individuals who chronically consume an average of 17 µg/day of shellfish, with a chromium intake maximum of 200 µg/person/day, the chromium level of concern would be 12 ppm. The maximum chromium level detected in muscle tissue in the Raritan River was 0.16 ppm which is significantly lower than 12 ppm. At the maximum chromium level detected in crab muscle tissue in the Raritan River, the corresponding chromium III intake is 2.7 µg/person/day which is significantly less than the maximum tolerable daily intake of 200 µg/person/day.

For lead, the document suggests a maximum tolerable daily intake of 25 µg/person/day. For individuals who chronically consume an average of 17 µg/day of shellfish, with a lead

intake maximum of 25 µg/person/day, the lead level of concern would be 1.5 ppm. The maximum lead level detected in crab muscle tissue in the Raritan River was 1.3 ppm which is below the lead level of concern of 1.5 ppm. At the maximum lead level detected in crab muscle tissue in the Raritan River, the corresponding lead intake is 22 µg/person/day which is below the maximum tolerable daily intake of 25 µg/person/day.

For nickel, the document suggests a maximum tolerable daily intake of 1200 µg/person/day. For individuals who chronically consume an average of 17 µg/day of shellfish, with a nickel intake maximum of 1200 µg/person/day, the nickel level of concern would be 70 ppm. The maximum lead level detected in crab muscle tissue in the Raritan River was 0.51 ppm which is below the nickel level of concern of 70 ppm. At the maximum nickel level detected in crab muscle tissue in the Raritan River, the corresponding nickel intake is 8.7 µg/person/day which is significantly less than the maximum tolerable daily intake of 1200 µg/person/day.

All maximum concentration of arsenic, cadmium, chromium III, lead and nickel detected in crab muscle tissue from the Raritan River were below the FDA levels of concern. See Table 2 for a comparison of the FDA Levels of Concern to the maximum daily intake of shellfish from AOC6-Raritan River.

Table 2: Comparison of FDA Levels of Concern to the Maximum Daily Intake of Shellfish from AOC6-Raritan River

Constituent	Level of Concern (µg/person-d)	Maximum Intake (µg/person-d)*
Arsenic	130	17
Cadmium	55	1.4
Chromium III	200	2.7
Lead	25	22
Nickel	1200	8.7

*Maximum intake of constituent by individuals who chronically consume an average of 17 µg/day of shellfish from AOC6 - Raritan River.

6.0 UNCERTAINTIES IN RISK ASSESSMENT

As in any risk assessment, the estimates of potential health threats (carcinogenic risks and noncarcinogenic health effects) for the Horseshoe Road Complex site have numerous associated uncertainties. The primary areas of uncertainty and limitations are qualitatively discussed here. In general, the main areas of uncertainty include the following:

- Environmental data
- Exposure pathway assumptions
- Toxicological data
- Risk characterization

Uncertainty is always involved in the estimation of chemical concentrations. Errors in the analytical data may stem from errors inherent in sampling and/or laboratory procedures. One of the most effective methods of minimizing procedural or systematic error is to subject the data to a strict quality control review. This quality control review procedure helps to eliminate many laboratory errors. However, even with all data vigorously validated, it must be realized that error is inherent in all laboratory procedures. Additional uncertainty occurred in this addendum HHRA because no duplicate samples were collected. This makes it difficult to validate the precision and accuracy of the samples.

The lack of site-specific exposure measurements requires that estimates be made on the basis of literature values and/or professional judgement. These types of estimates were required in the evaluation of exposure scenario input parameters. For example, assumptions were made for the exposure time, frequency, and duration of potential chemical exposures as well as for the quantity of ingested chemical contaminants. In general, assumptions were made based on reasonable maximum exposures.

Other standard assumptions used throughout this risk assessment are assumed to represent average values (i.e., 70 kg average adult body weight) or upper-bounds of potential exposure and have been used as appropriate.

Toxicological data uncertainty is one of the largest sources of error in this risk assessment. Numerous uncertainties are associated with USEPA-derived toxicity values used in risk assessment. One source of uncertainty may include using dose-response information from effects observed at high doses in animals to predict adverse health effects from low level exposures to humans in contact with the chemical in the environment. Another source may be the use of dose-response information from short-term exposure studies to predict the effects of long-term exposure and vice versa. Uncertainties may also arise from using dose-

response information in animals to predict human health effects and from homogeneous animal and healthy human populations to predict effects likely to be observed in the general population which consists of individuals with varying sensitivities. In addition, the inability to quantitatively evaluate all chemicals detected at the site due to the lack of sufficient toxicological data may result in underestimation of risks and/or health effects. Chemicals without toxicity data include copper, lead, mercury, and dimethyl phthalate. These four COCs are qualitatively evaluated in Section 5.0 Risk Characterization.

Other toxicological data uncertainty in this risk assessment includes the use of the benzo(a)pyrene oral slope factor in conjunction with relative potency values to develop slope factors for 1,2-Benzphenanthracene (Chrysene), the combining of carcinogens with different weights-of-evidence in the calculation of risk; and the combining of noncarcinogens with different toxicity endpoints in the calculation of hazard index values.

Additionally, because shellfish are migratory in nature, the contaminants present in the crab tissue may have been derived from other areas. In the future, NJDEP's Toxics in Biota Monitoring Program will have crab tissue data available for identifying background levels in shellfish in the Raritan River. These data will enable us to determine whether contamination in the shellfish is comparable to other areas of the river.

As a result of the uncertainties described above, this risk assessment should not be construed as presenting absolute risks or hazards. Rather, it is a conservative analysis intended to indicate the potential for adverse impacts to occur, based on a reasonable maximum exposure.

7.0 SUMMARY OF THE ADDENDUM RISK ASSESSMENT

In this addendum Human Health Risk Assessment, shellfish at the Horseshoe Road Complex site were quantitatively evaluated for potential health threats to human receptors via the ingestion pathway. Adult residents were evaluated under present and potential future land use conditions, as appropriate. The estimates of risk and hazard and the greatest chemical contributors to these estimates have been presented and discussed.

Chemicals of potential concern were selected based on criteria outlined in RAGS (USEPA, 1989) and are presented in Appendix D. The chemicals of potential concern included VOCs, SVOCs, pesticides, and inorganics. The essential nutrients (i.e., calcium, magnesium, potassium, and sodium) were not quantitatively addressed as their potential toxicity is significantly lower than other inorganics at the site, and most existing toxicological data pertain to dietary intake.

Exposure routes and human receptor groups were identified and quantitative estimates of the magnitude, frequency, and duration of exposure were made. Exposure points were estimated using the maximum concentration. Chronic daily intakes for the ingestion route was calculated for the reasonable maximum exposure (i.e., using maximum concentrations and the 90^{th} and 95^{th} percentile exposure parameters).

In the toxicity assessment, current toxicological human health data (i.e., reference doses and slope factors) were obtained from various sources and were utilized in the order as specified by RAGS (USEPA, 1989a). Toxicological profiles for the chemicals of potential concern have been developed and were presented in Appendix E of the baseline HHRA (CDM Federal, 1999a) and of this HHRA Addendum.

Risk characterization involved integrating the exposure and toxicity assessments into quantitative expressions of risks/health effects. Specifically, chronic daily intakes were compared with concentrations known or suspected to present health risks or hazards. The carcinogenic risks and noncarcinogenic hazard index values calculated for the site are based on the reasonable maximum exposure (the highest exposure reasonably expected to occur at a site). The intent is to estimate a conservative exposure case that is still within the range of possible exposures.

In accordance with the National Oil and Hazardous Substance Pollution Contingency Plan (NCP) Section 300.430 (e)(2) for known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper-bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6}. Per RAGS Part B: Development of

Risk-Based Preliminary Remediation Goals (USEPA, 1991b), for noncarcinogenic effects, the NCP does not specify a range, but it is generally appropriate to assume a hazard index equal to 1.

In general, the USEPA recommends target values or ranges (i.e., risk of 10^{-4} to 10^{-6} or hazard index of one) as threshold values for potential human health impacts (USEPA, 1989a). These target values aid in determining the objectives of the baseline human health risk assessment which include determining whether additional response action is necessary at the site, by providing a basis for determining residual chemical levels that are adequately protective of human health, by providing a basis for comparing potential health impacts of various remedial alternatives, and to help support selection of the "no action" remedial alternative, where appropriate.

In summary, a review of the carcinogenic risks and noncarcinogenic hazards for the ingestion of shellfish from the Raritan River by the adult resident receptor showed values that fell within the USEPA's target risk range of 10^{-4} to 10^{-6} and below a hazard index of 1. The overall carcinogenic risks and noncarcinogenic hazards for the shellfish, surface water and sediment media together, showed an exceedance of USEPA's target risk range and hazard index.

Site-specific uncertainties relating to the risk assessment were qualitatively addressed in Section 6.0. Because no carcinogenic risks or noncarcinogenic hazards were above the USEPA's target risk range for the ingestion of shellfish, central tendency calculations were not performed in the addendum HHRA as a quantitative measure of uncertainty in the risk assessment.

8.0 REFERENCES

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National Contingency Plan (NCP). Volume 40 Code of Federal Regulations (CFR) Part 300.

FIGURES

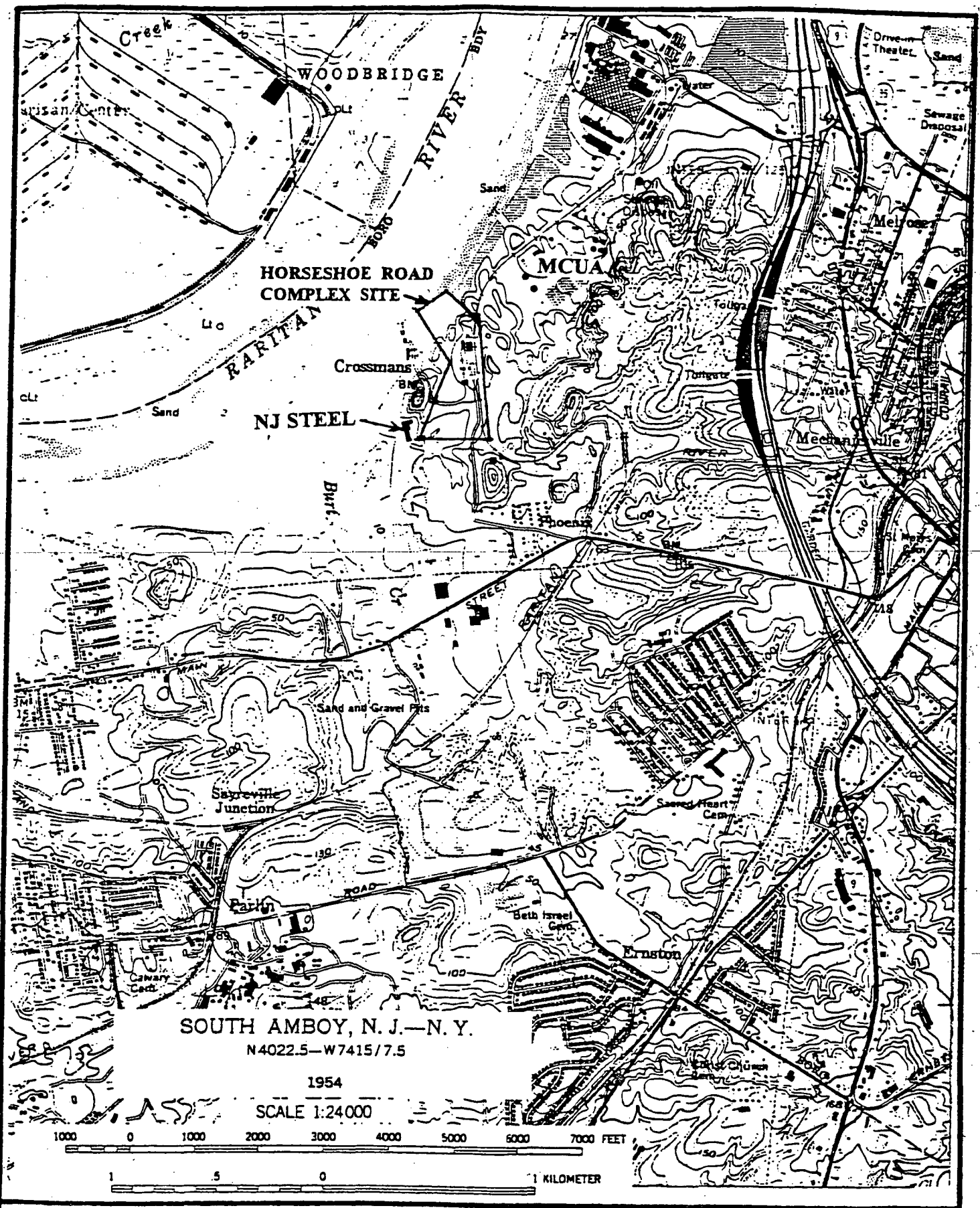


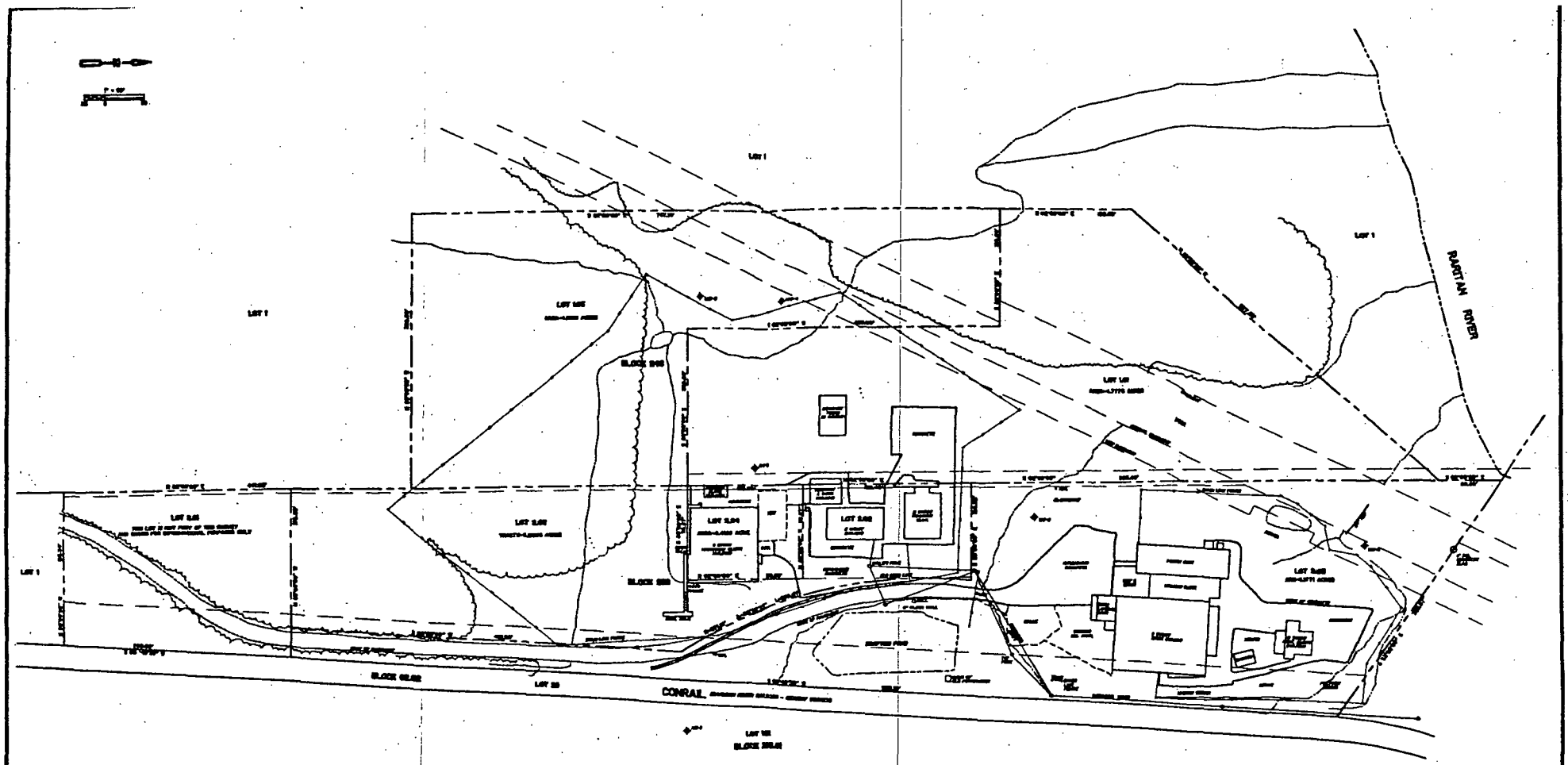
FIGURE 1

HORSESHOE ROAD SITE LOCATION MAP

HORSESHOE ROAD COMPLEX SITE
SAYREVILLE, NEW JERSEY
WORK ASSIGNMENT 013-RICO-02BT

CDM FEDERAL PROGRAMS CORPORATION
a subsidiary of Camp Dresser & McKee Inc.

400347



REFERENCE, LOCATION SURVEY FOR ALLIANCE TECHNOLOGIES CORPORATION, SITUATED IN THE BOROUGH OF SAYREVILLE, MIDDLESEX COUNTY, NEW JERSEY, DATED MARCH 11, 1992.

NOTE, NOT TO SCALE

FIGURE 2

SITE MAP

HORSESHOE ROAD COMPLEX SITE
SAYREVILLE, NEW JERSEY
WORK ASSIGNMENT 013-RICO-02BT

CDM FEDERAL PROGRAMS CORPORATION
a subsidiary of Camp Dresser & McKee Inc.

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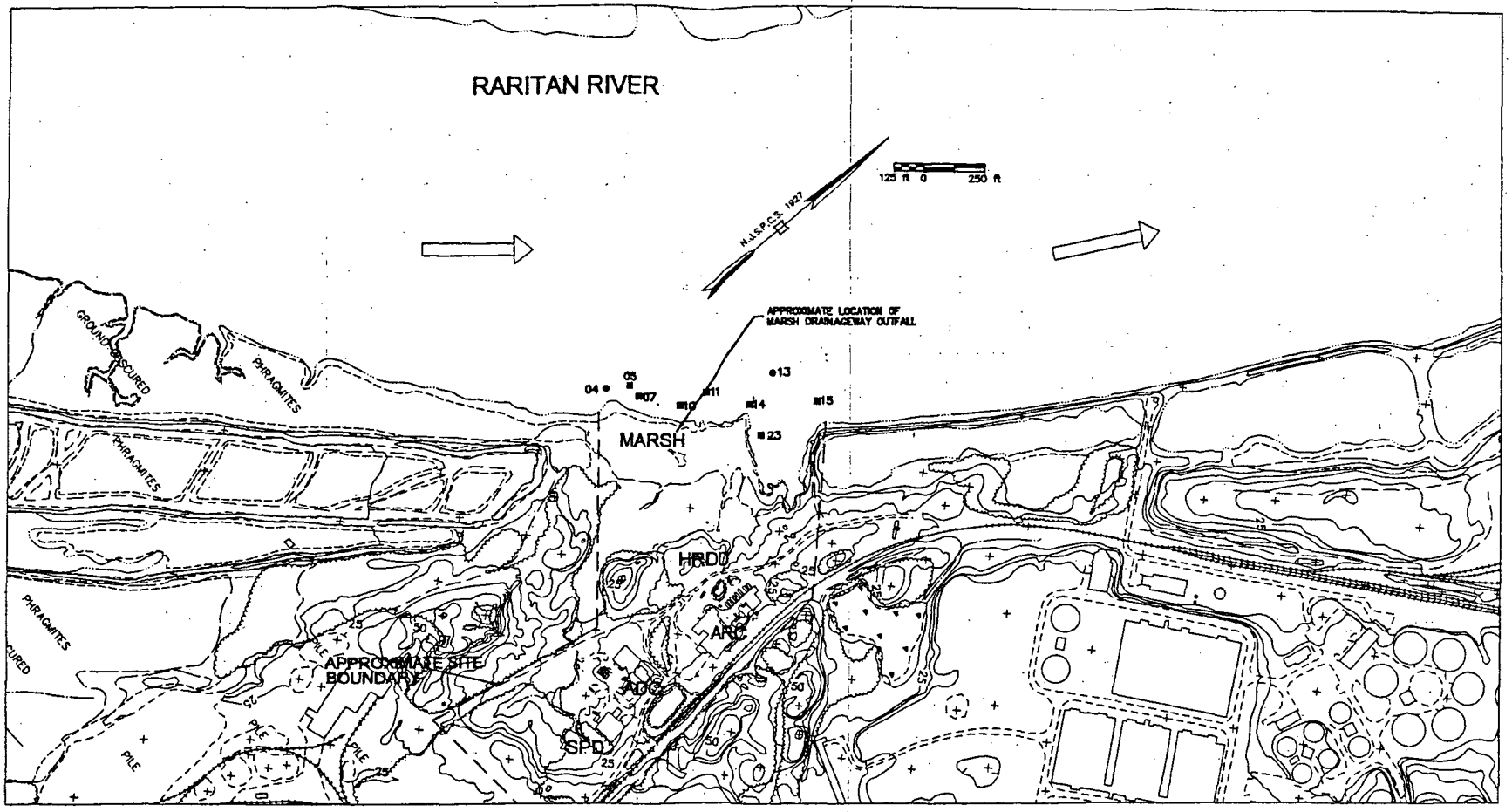

Contour Interval = 5 feet above mean sea level

LEGEND

HRDD	HORSESHOE ROAD DRUM DUMP	■05	BIOTA
ARC	ATLANTIC RESOURCES CORPORATION	●13.	BIOTA AND TOXICITY
ADC	ATLANTIC DEVELOPMENT CORPORATION		
SPD	SAYREVILLE PESTICIDE DUMP		
⇨	DOMINANT STREAMFLOW DIRECTION		

NEW NO.	DATE	DRWN	CHKD	REMARKS

AG
A subsidiary of Camp Dresser & McKee Inc.

HORSESHOE ROAD COMPLEX SITE
SAYREVILLE, NEW JERSEY

HORSESHOE ROAD COMPLEX SITE

FIGURE 3

BIOTA AND TOXICITY SAMPLING LOCATIONS

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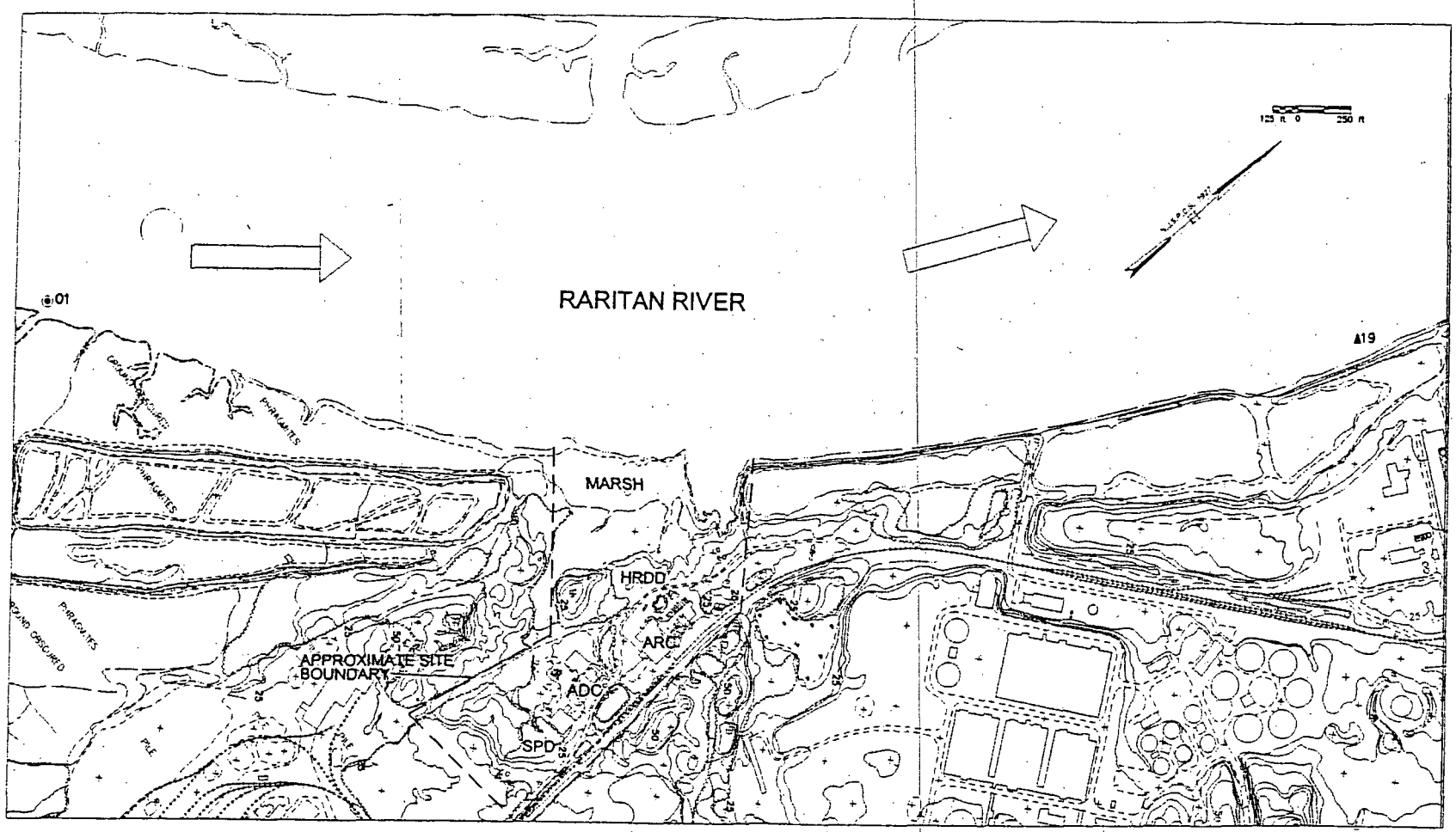

LEGEND

HRDD	HORSESHOE ROAD DRUM DUMP	◉01	BIOTA
ARC	ATLANTIC RESOURCES CORPORATION	■02	TOXICITY
ADC	ATLANTIC DEVELOPMENT CORPORATION	●18	SURFACE WATER/ SEDIMENT ONLY (RSW##RSD##)
SPD	SAYREVILLE PESTICIDE DUMP	▲19	BIOTA AND DIOXIN

NOTE: SURFACE WATER/SEDIMENT SAMPLES WERE TAKEN AT EVERY RARITAN RIVER LOCATION.

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CDM Federal Programs Corporation
A subsidiary of Camp Dresser & McKee Inc.

HORSESHOE ROAD COMPLEX SITE
SAYREVILLE, NEW JERSEY

HORSESHOE ROAD SUPERFUND SITE

FIGURE 4

REFERENCE SAMPLING LOCATIONS

Epcal\Temp\Maysill\Horseshoe\reference-sampling-loc.dwg Fri Jul 14 16:44:47 2000 sullivan

APPENDIX A
STANDARD TABLES

TABLE 1

SELECTION OF EXPOSURE PATHWAYS

HORSESHOE ROAD COMPLEX, SAYREVILLE, NEW JERSEY

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Surface Water	Shellfish	Raritan River	Residents	Adult	Ingestion	On-Site	Quant	Residents may ingest shellfish caught in the Raritan River that have been potentially impacted by site contaminants released into surface water.

Quant = Quantitative risk analysis performed. Qual=Qualitative analysis performed.

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TABLE 2
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
HORSESHOE ROAD COMPLEX, SAYREVILLE, NEW JERSEY

Scenario Timeframe: *Current/Future*
Medium: Surface Water
Exposure Medium: Shellfish
Exposure Point: Raritan River

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency		Range of Detection Limits	Concentration Used for Screening	Background Value (3)	Screening (4) Toxicity Value		Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (5) Contaminant Deletion or Selection
7429-90-5	Aluminum	3.7		6	B	mg/kg	RCM04	7/	7	N/A	6	3.5	1.40E+03	N	N/A	N/A	No	BTX
7440-36-0	Antimony	0.08	BJ	0.14	BJ	mg/kg	RCM14	4/	7	.07-.07	0.14	0.11	5.40E-01	N	N/A	N/A	Yes	TX
7440-38-2	Arsenic	0.48		1	J	mg/kg	RCM15	9/	9	N/A	1	0.72	2.10E-03	C	N/A	N/A	Yes	TX
7440-39-3	Barium	0.11	B	0.58	B	mg/kg	RCM11	9/	9	N/A	0.58	0.3	9.50E+01	N	N/A	N/A	No	BTX
7440-41-7	Beryllium	0.02		0.04	B	mg/kg	RCM05	3/	9	.02-.02	0.04	0.01	2.70E+00	N	N/A	N/A	No	BTX
7440-43-9	Cadmium	0.03	B	0.08	B	mg/kg	RCM14	4/	9	.02-.02	0.08	0.06	1.40E+00	N	N/A	N/A	Yes	TX
7440-70-2	Calcium Metal	453	J	2700	J	mg/kg	RCM11	9/	9	N/A	2700	1077	NA	NA	N/A	N/A	No	NUT
16065-83-1	Chromium III	0.07	B	0.16	B	mg/kg	RCM11	9/	9	N/A	0.16	0.10	2.00E+03	N	N/A	N/A	No	BTX
18540-29-9	Chromium VI	0.01	B	0.03	B	mg/kg	RCM11	9/	9	N/A	0.03	0.02	4.10E+00	N	N/A	N/A	No	BTX
7440-50-8	Copper	11.4		13.6		mg/kg	RCM07	4/	4	N/A	13.6	17.3	5.40E+01	N	N/A	N/A	No	NTX
7439-89-6	Iron	2.7	BE*	18.1	J	mg/kg	RCM05	5/	5	N/A	18.1	8.1	4.10E+02	N	N/A	N/A	No	NUT
7439-92-1	Lead	0.42		1.3	*	mg/kg	RCM10	5/	7	0.86-0.92	1.3	0.90	NA	NA	N/A	N/A	No	NTX
7439-95-4	Magnesium	279	J	424	J	mg/kg	RCM11	9/	9	N/A	424	357	NA	NA	N/A	N/A	No	NUT
7439-96-5	Manganese	0.58	BE*	2.3	J	mg/kg	RCM11	5/	5	N/A	2.3	1.1	1.90E+02	N	N/A	N/A	No	BTX
7439-97-6	Mercury	0.04		0.05	M	mg/kg	RCM04	2/	9	.03-.03	0.05	0.02	NA	NA	N/A	N/A	No	NTX
7440-02-0	Nickel	0.07	BJ	0.51	BJ	mg/kg	RCM10	5/	9	.04-.04	0.51	0.04	2.70E+01	N	N/A	N/A	No	BTX
7440-09-7	Potassium	1710		2620	J	mg/kg	RCM13	9/	9	N/A	2620	2390	NA	NA	N/A	N/A	No	NUT
7782-49-2	Selenium	0.4	NJ	1.3	*J	mg/kg	RCM15	9/	9	N/A	1.3	0.73	6.80E+00	N	N/A	N/A	Yes	TX
7440-22-4	Silver	0.05	BNJ	0.55		mg/kg	RCM15	6/	9	.02-.02	0.55	0.15	6.80E+00	N	N/A	N/A	Yes	TX
7440-23-5	Sodium	2680	J	5590		mg/kg	RCM05	9/	9	N/A	5590	4215	NA	NA	N/A	N/A	No	NUT
7440-66-6	Zinc	26.7	J	49.7	J	mg/kg	RCM11	9/	9	N/A	49.7	35.5	4.10E+02	N	N/A	N/A	Yes	TX
218-01-9	1,2-Benzphenanthracene (Chrysene)	53	J	53	J	ug/kg	RCM23	1/	9	330-330	53	165	4.30E+02	C	N/A	N/A	No	BTX
78-93-3	2-Butanone	2	J	13	J	ug/kg	RCM07	4/	9	10-10	13	4	8.10E+05	N	N/A	N/A	No	BTX
95-48-7	2-Methylphenol	34	J	34	J	ug/kg	RCM10	1/	9	330-330	34	165	6.80E+04	N	N/A	N/A	No	BTX
72-54-8	4,4'-DDD	11	JN	110		ug/kg	RCM05	3/	9	5-5	110	3.9	1.30E+01	C	N/A	N/A	Yes	TX
72-55-9	4,4'-DDE	8.4	J	120		ug/kg	RCM05	5/	9	5-5	120	2.5	9.30E+00	C	N/A	N/A	Yes	TX
67-64-1	Acetone	13		140		ug/kg	RCM23	8/	9	10-10	140	72.5	1.40E+05	N	N/A	N/A	No	BTX
117-81-7	bis(2-Ethylhexyl) Phthalate	52	J	320	J	ug/kg	RCM10	8/	9	330-330	320	133	2.30E+02	C	N/A	N/A	Yes	TX
75-15-0	Carbon Disulfide	7	J	15		ug/kg	RCM13	6/	9	10-10	15	10.5	1.40E+05	N	N/A	N/A	No	BTX
75-09-2	Dichloromethane	2	J	2	J	ug/kg	RCM04 (2)	3/	9	10-10	2	3.5	4.20E+02	C	N/A	N/A	No	BTX
60-57-1	Dieldrin	9.7		9.7		ug/kg	RCM05	1/	9	5-5	9.7	2.5	2.00E-01	C	N/A	N/A	Yes	TX
131-11-3	Dimethyl phthalate	35	J	35	J	ug/kg	RCM07	1/	9	330-330	35	165	1.40E+07	N	N/A	N/A	No	NTX
84-74-2	Di-n-Butyl Phthalate	72	J	390		ug/kg	RCM10	6/	9	270-330	390	157.5	1.40E+05	N	N/A	N/A	No	BTX
33213-65-9	Endosulfan II	4.5	J	11	J	ug/kg	RCM14	2/	9	5-5	11	2.5	8.10E+03	N	N/A	N/A	No	BTX
1024-57-3	Heptachlor Epoxide	8.7	J	21	P	ug/kg	RCM05	3/	9	5-5	21	2.5	3.50E-01	C	N/A	N/A	Yes	TX
129-00-0	Pyrene	51	J	51	J	ug/kg	RCM04	1/	9	330-330	51	165	4.10E+04	N	N/A	N/A	No	BTX
1330-20-7	Xylenes, Total	2	J	2	J	ug/kg	RCM05	1/	9	10-10	2	5	2.70E+06	N	N/A	N/A	No	BTX

(1) Minimum/maximum detected concentration.
(2) Maximum concentration detected at RCM04, RCM07 and RCM23.
(3) Shellfish samples RCM01 and RCM19 were used for background.
(4) Region III Risk-Based Concentration for fish were used as toxicity screen.
(5) Rationale Codes
Selection Reason: Toxicity Information Available (TX)
Special Case (SC)
Deletion Reason: Background Levels (BKG)
No Toxicity Information (NTX)
Essential Nutrient (NUT)
Frequency of Detection < 1% (FRQ)
Below Concentration Toxicity Screen of 1% (BTX)

Definitions: N/A = Not Applicable
NA = Not Available
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
C = Carcinogenic
N = Non-Carcinogenic
B = Reported value is <CRDL, but >IDL
E = Value is estimated because of the presence of interference.
M = Duplicate injection precision not met
N (Inorganic) = Sample recovery is not within control limits
* = Duplicate analysis not within control limits
J = Estimated data due to exceeded quality control criteria
N (Organic) = Presumptive evidence of a compound
P = The difference for detected conc. of a pesticide is >25% between the two GC columns.

TABLE 3
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
HORSESHOE ROAD COMPLEX SITE, SAYREVILLE, NEW JERSEY

Scenario Timeframe: Current and Future
Medium: Surface Water
Exposure Medium: Shellfish
Exposure Point: AOC 6 - RR

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier (4)	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
Aluminum (Fume or Dust)	mg/kg	4.87	N/A (3)	6	B	mg/kg	6	Max	(1)	4.87	Mean-N	(2)
Antimony	mg/kg	0.11	N/A (3)	0.14	BJ	mg/kg	0.14	Max	(1)	0.11	Mean-N	(2)
Arsenic	mg/kg	0.73	N/A (3)	1	J	mg/kg	1	Max	(1)	0.73	Mean-N	(2)
Barium	mg/kg	0.23	N/A (3)	0.58	B	mg/kg	0.58	Max	(1)	0.23	Mean-N	(2)
Beryllium	mg/kg	0.03	N/A (3)	0.04	B	mg/kg	0.04	Max	(1)	0.03	Mean-N	(2)
Cadmium	mg/kg	0.05	N/A (3)	0.08	B	mg/kg	0.08	Max	(1)	0.05	Mean-N	(2)
Calcium Metal	mg/kg	1189	N/A (3)	2700	J	mg/kg	2700	Max	(1)	1189	Mean-N	(2)
Chromium III	mg/kg	0.10	N/A (3)	0.16	B	mg/kg	0.16	Max	(1)	0.10	Mean-N	(2)
Chromium VI	mg/kg	0.02	N/A (3)	0.03	B	mg/kg	0.03	Max	(1)	0.02	Mean-N	(2)
Copper	mg/kg	12.6	N/A (3)	13.6		mg/kg	13.6	Max	(1)	12.6	Mean-N	(2)
Iron	mg/kg	9.38	N/A (3)	18.1	J	mg/kg	18.1	Max	(1)	9.38	Mean-N	(2)
Lead	mg/kg	0.62	N/A (3)	1.3	*	mg/kg	1.3	Max	(1)	0.62	Mean-N	(2)
Magnesium	mg/kg	347	N/A (3)	424	J	mg/kg	424	Max	(1)	347	Mean-N	(2)
Manganese	mg/kg	1.07	N/A (3)	2.3	J	mg/kg	2.3	Max	(1)	1.07	Mean-N	(2)
Mercury	mg/kg	0.05	N/A (3)	0.05	M	mg/kg	0.05	Max	(1)	0.05	Mean-N	(2)
Nickel	mg/kg	0.2	N/A (3)	0.51	BJ	mg/kg	0.51	Max	(1)	0.2	Mean-N	(2)
Potassium	mg/kg	2196	N/A (3)	2620	J	mg/kg	2620	Max	(1)	2196	Mean-N	(2)
Selenium	mg/kg	0.65	N/A (3)	1.3	*J	mg/kg	1.3	Max	(1)	0.65	Mean-N	(2)
Silver	mg/kg	0.28	N/A (3)	0.55		mg/kg	0.55	Max	(1)	0.28	Mean-N	(2)
Sodium	mg/kg	3909	N/A (3)	5590		mg/kg	5590	Max	(1)	3909	Mean-N	(2)
Zinc	mg/kg	38.5	N/A (3)	49.7	J	mg/kg	49.7	Max	(1)	38.5	Mean-N	(2)
1,2-Benzphenanthracene (Chrysene)	ug/kg	53	N/A (3)	53	J	ug/kg	53	Max	(1)	53	Mean-N	(2)
2-Butenone	ug/kg	5	N/A (3)	13	J	ug/kg	13	Max	(1)	5	Mean-N	(2)
2-Methylphenol	ug/kg	34	N/A (3)	34	J	ug/kg	34	Max	(1)	34	Mean-N	(2)
4,4'-DDD	ug/kg	46.3	N/A (3)	110		ug/kg	110	Max	(1)	46.3	Mean-N	(2)
4,4'-DDE	ug/kg	35.9	N/A (3)	120		ug/kg	120	Max	(1)	35.9	Mean-N	(2)
Acetone	ug/kg	80.0	N/A (3)	140		ug/kg	140	Max	(1)	80.0	Mean-N	(2)
bis(2-Ethylhexyl) Phthalate	ug/kg	181	N/A (3)	320	J	ug/kg	320	Max	(1)	181	Mean-N	(2)
Carbon Disulfide	ug/kg	9.17	N/A (3)	15		ug/kg	15	Max	(1)	9.17	Mean-N	(2)
Dichloromethane	ug/kg	2	N/A (3)	2	J	ug/kg	2	Max	(1)	2	Mean-N	(2)
Dieldrin	ug/kg	9.7	N/A (3)	9.7		ug/kg	9.7	Max	(1)	9.7	Mean-N	(2)
Dimethyl Phthalate	ug/kg	35	N/A (3)	35	J	ug/kg	35	Max	(1)	35	Mean-N	(2)
Di-n-Butyl Phthalate	ug/kg	203	N/A (3)	390		ug/kg	390	Max	(1)	203	Mean-N	(2)
Endosulfan II	ug/kg	7.75	N/A (3)	11	J	ug/kg	11	Max	(1)	7.75	Mean-N	(2)
Heptachlor Epoxide	ug/kg	13.0	N/A (3)	21	P	ug/kg	21	Max	(1)	13.0	Mean-N	(2)
Pyrene	ug/kg	51	N/A (3)	51	J	ug/kg	51	Max	(1)	51	Mean-N	(2)
Xylenes, Total	ug/kg	2	N/A (3)	2	J	ug/kg	2	Max	(1)	2	Mean-N	(2)

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N).

N/A - Not Applicable.

(1) 95% UCL is not applicable because there are fewer than 10 samples. Therefore, maximum concentration used for EPC.

(2) 95% UCL is not applicable because there are fewer than 10 samples. Therefore, arithmetic average concentration used for EPC.

(3) 95% UCL is not applicable because there are fewer than 10 samples.

(4) Definitions of the qualifiers may be found in the HHRA Addendum Document.

TABLE 4
VALUES USED FOR DAILY INTAKE CALCULATIONS
HORSESHOE ROAD COMPLEX, SAYREVILLE, NEW JERSEY

Scenario Timeframe: Current and Future
Medium: Surface Water
Exposure Medium: Shellfish
Exposure Point: Raritan River
Receptor Population: Residents
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion of Shellfish	CSF	Chemical Concentration in Shellfish	mg/kg	Chem.-specific Max.*	-	Chem.-specific Average		Chronic Daily Intake (CDI) (mg/kg/day) = CSF x IR x EF x ED x 1/BW x 1/AT
	IR	Ingestion rate	kg/day	0.0065	RAGS, Part A			RME
	EF	Exposure Frequency	days/yr	350	RAGS, Part A			CDI = CSF x 8.9E-5 (Noncarcinogenic)
	ED	Exposure Duration	yrs	24	RAGS, Part A	9	RAGS, Part A	CDI = CSF x 3.1E-5 (Carcinogenic)
	BW	Body Weight	kg	70	RAGS, Part A			CT
	AT-NC	Averaging Time (noncancer)	days	8,760	RAGS, Part A	3,285	RAGS, Part A	CDI = CSF x 8.9E-5 (Noncarcinogenic)
	AT- C	Averaging Time (cancer)	days	25,550	RAGS, Part A			CDI = CSF x 1.1E-5 (Carcinogenic)

References:
RAGS, Part A. US EPA, Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual, Part A, Interim Final. December 1989.

Notes:
* - The maximum concentration will be used because the sample size is nine samples.

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10/31/00

TABLE 5
NON-CANCER CHRONIC TOXICITY DATA -- ORAL
HORSESHOE ROAD COMPLEX SITE, SAYREVILLE, NEW JERSEY

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD Value	Oral RfD Units	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfD: Target Organ	Dates of RfD: Target Organ (MM/DD/YY)
Volatile Organics							
Acetone	Chronic	1.0E-01	mg/kg/day	Liver/kidney	1000	IRIS (1)	8/24/00
2-Butanone	Chronic	6.0E-01	mg/kg/day	Fetus	3000	IRIS	8/24/00
Carbon Disulfide	Chronic	1.0E-01	mg/kg/day	Fetus	100	IRIS	8/24/00
Methylene Chloride	Chronic	6.0E-02	mg/kg/day	Liver	100	IRIS	8/24/00
Xylenes (Total)*	Chronic	2.0E+00	mg/kg/day	CNS/Whole Body	100	IRIS	8/24/00
Semivolatile Organics							
1,2-Benzphenanthracene (Chrysene)*	Chronic	-	mg/kg/day	-	-	-	-
Bis(2-ethylhexyl)phthalate	Chronic	2.0E-02	mg/kg/day	Liver	1000	IRIS	8/24/00
Di-n-butyl phthalate	Chronic	1.0E-01	mg/kg/day	Whole Body	1000	IRIS	8/24/00
Dimethylphthalate	Chronic	-	mg/kg/day	-	-	-	-
2-Methylphenol	Chronic	5.0E-02	mg/kg/day	Whole Body/CNS	1000	IRIS	8/24/00
Pyrene	Chronic	3.0E-02	mg/kg/day	Kidney	3000	IRIS	8/24/00
Pesticides/PCBs							
4,4'-DDD	Chronic	-	mg/kg/day	-	-	-	-
4,4'-DDE	Chronic	-	mg/kg/day	-	-	-	-
Dieldrin	Chronic	5.0E-05	mg/kg/day	Liver	100	IRIS	8/24/00
Endosulfan II	Chronic	6.0E-03	mg/kg/day	Whole Body/Kidney/Liver	100	IRIS (2)	8/24/00
Heptachlor Epoxide	Chronic	1.3E-05	mg/kg/day	Liver	1000	IRIS	8/24/00
Inorganics							
Aluminum	Chronic	1.0E+00	mg/kg/day	GI Tract/CNS	100	NCEA (3)	9/12/00
Antimony	Chronic	4.0E-04	mg/kg/day	Whole Body/Blood	1000	IRIS	8/24/00
Arsenic	Chronic	3.0E-04	mg/kg/day	Skin	3	IRIS	8/24/00
Barium	Chronic	7.0E-02	mg/kg/day	Cardiovascular	3	IRIS	8/24/00
Beryllium	Chronic	2.0E-03	mg/kg/day	Small Intestine	300	IRIS	8/24/00
Cadmium (food)	Chronic	1.0E-03	mg/kg/day	Kidney	10	IRIS	8/24/00
Chromium III (insoluble salts)	Chronic	1.5E+00	mg/kg/day	Lung	100/10	IRIS	8/24/00
Chromium VI	Chronic	3.0E-03	mg/kg/day	Stomach/Intestine	300/3	IRIS	8/24/00
Copper	Chronic	-	mg/kg/day	-	-	-	-
Lead (and compounds-inorg.)**	Chronic	-	mg/kg/day	-	-	-	-
Manganese	Chronic	1.4E-01	mg/kg/day	CNS, Ingestion	1	IRIS	8/30/00
Mercury (elemental)	Chronic	-	mg/kg/day	-	-	-	-
Nickel (soluble salt)	Chronic	2.0E-02	mg/kg/day	Whole Body Organs	300	IRIS	8/24/00
Selenium	Chronic	5.0E-03	mg/kg/day	Liver	3	IRIS	8/24/00
Silver	Chronic	5.0E-03	mg/kg/day	Skin	3	IRIS	8/24/00
Zinc (and compounds)	Chronic	3.0E-01	mg/kg/day	Blood	3	IRIS	8/24/00

Notes:
- Calcium, iron, magnesium, potassium, and sodium are considered essential nutrients and will not be quantitatively evaluated in the risk assessment.
* - Relative potency values were used in conjunction with the benzo(a)pyrene oral slope factor per NCEA guidance 9/12/00.
** - Since no noncarcinogenic toxicity values are currently established for lead, only a qualitative evaluation of this chemical can be performed.
(1) All toxicity values were obtained from Integrated Risk Information System (IRIS) (on-line August 2000) unless otherwise noted.
(2) The noncarcinogenic toxicity values for endosulfan are reported from IRIS, as the individual endosulfan I and endosulfan II isomers do not have established noncarcinogenic toxicity values.

TABLE 6
CANCER TOXICITY DATA -- ORAL
HORSESHOE ROAD COMPLEX SITE, SAYREVILLE, NEW JERSEY

Chemical of Potential Concern	Oral Cancer Slope Factor	Units	Weight of Evidence/ Cancer Guideline Description	Source	Date (MM/DD/YY)
Volatile Organics					
Acetone	-	-	D	-	-
2-Butanone	-	-	D	-	-
Carbon Disulfide	-	-	-	-	-
Methylene Chloride	7.5E-03	(mg/kg/day)-1	B2	IRIS (1)	08/24/00
Xylenes (Total)	-	-	D	-	-
Semivolatile Organics					
1,2-Benzphenanthracene (Chrysene)*	7.3E-03	(mg/kg/day)-1	B2	NCEA (2)	09/12/00
Bis(2-ethylhexyl)phthalate	1.4E-02	(mg/kg/day)-1	B2	IRIS	08/24/00
Di-n-butyl phthalate	-	-	D	-	-
Dimethylphthalate	-	-	D	-	-
2-Methylphenol	-	-	C	-	-
Pyrene	-	-	D	-	-
Pesticides/PCBs					
4,4'-DDD	2.4E-01	(mg/kg/day)-1	B2	IRIS	08/24/00
4,4'-DDE	3.4E-01	(mg/kg/day)-1	B2	IRIS	08/24/00
Dieldrin	1.6E+01	(mg/kg/day)-1	B2	IRIS	08/24/00
Endosulfan II	-	-	-	(3)	-
Heptachlor Epoxide	9.1E+00	(mg/kg/day)-1	B2	IRIS	08/24/00
Inorganics					
Aluminum	-	-	-	-	-
Antimony	-	-	-	-	-
Arsenic	1.5E+00	(mg/kg/day)-1	A	IRIS	08/24/00
Barium	-	-	-	-	-
Beryllium	-	-	B1	-	-
Cadmium	-	-	B1	-	-
Chromium III (insolublesalts)	-	-	-	-	-
Chromium VI	-	-	D	-	-
Copper	-	-	D	-	-
Lead (and compounds-inorg.)**	-	-	B2	-	-
Manganese	-	-	D	-	-
Mercury	-	-	D	-	-
Nickel (soluble salt)	-	-	-	-	-
Selenium (and compounds)	-	-	D	-	-
Silver	-	-	D	-	-
Zinc (and compounds)	-	-	D	-	-

Notes:

- Calcium, iron, magnesium, potassium, and sodium are considered essential nutrients and will not be quantitatively evaluated in the risk assessment.
* Relative potency values were used in conjunction with the benzo(a)pyrene oral slope factor per NCEA guidance 9/12/00.
**Since no carcinogenic toxicity values are currently established for lead, only a qualitative evaluation of this chemical can be performed.
(1) All toxicity values were obtained from IRIS (on-line August 2000) unless otherwise noted.
(2) Toxicity values were obtained from the National Center for Environmental Assessment (NCEA) September12, 2000.
(3) No carcinogenic toxicity values are currently established for endosulfan or its isomers endosulfan I and endosulfan II.

EPA Group:
- A - Human carcinogen
- B1 - Probable human carcinogen - indicates that limited human data are available
- B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans
- C - Possible human carcinogen
- D - Not classifiable as a human carcinogen
- E - Evidence of noncarcinogenicity

Weight of Evidence:
- Known/Likely
- Cannot be Determined
- Not Likely

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TABLE 7 RME
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
HORSESHOE ROAD COMPLEX SITE, SAYREVILLE, NEW JERSEY

Scenario Timeframe: Current and Future
Medium: Surface Water
Exposure Medium: Shellfish
Exposure Point: AOC 6 - RR
Receptor Population: Residents
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose (2)	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	Antimony	0.14	mg/kg	0.14	mg/kg	M	1.2E-05	mg/kg-day	4.0E-04	mg/kg-day	N/A	N/A	3.1E-02
	Arsenic	1	mg/kg	1	mg/kg	M	8.9E-05	mg/kg-day	3.0E-04	mg/kg-day	N/A	N/A	3.0E-01
	Cadmium	0.08	mg/kg	0.08	mg/kg	M	7.1E-06	mg/kg-day	1.0E-03	mg/kg-day	N/A	N/A	7.1E-03
	Selenium	1.3	mg/kg	1.3	mg/kg	M	1.2E-04	mg/kg-day	5.0E-03	mg/kg-day	N/A	N/A	2.3E-02
	Silver	0.55	mg/kg	0.55	mg/kg	M	4.9E-05	mg/kg-day	5.0E-03	mg/kg-day	N/A	N/A	9.8E-03
	Zinc	49.7	mg/kg	49.7	mg/kg	M	4.4E-03	mg/kg-day	3.0E-01	mg/kg-day	N/A	N/A	1.5E-02
	4,4'-DDD	110	ug/kg	110	ug/kg	M	9.8E-06	mg/kg-day	--	mg/kg-day	N/A	N/A	--
	4,4'-DDE	120	ug/kg	120	ug/kg	M	1.1E-05	mg/kg-day	--	mg/kg-day	N/A	N/A	--
	bis(2-Ethylhexyl) Phthalate	320	ug/kg	320	ug/kg	M	2.8E-05	mg/kg-day	2.0E-02	mg/kg-day	N/A	N/A	1.4E-03
	Dieldrin	9.7	ug/kg	9.7	ug/kg	M	8.6E-07	mg/kg-day	5.0E-05	mg/kg-day	N/A	N/A	1.7E-02
	Heptachlor Epoxide	21	ug/kg	21	ug/kg	M	1.9E-06	mg/kg-day	1.3E-05	mg/kg-day	N/A	N/A	1.4E-01
												Total Hazard Index	5.5E-01

(1) Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.
(2) Chronic.

-- - Reference Dose not available, therefore Hazard Quotient not calculated.
N/A - Not Applicable.

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10/31/00

TABLE 8 RME
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
HORSESHOE ROAD COMPLEX SITE, SAYREVILLE, NEW JERSEY

Scenario Timeframe: Current and Future
Medium: Surface Water
Exposure Medium: Shellfish
Exposure Point: AOC 6 - RR
Receptor Population: Residents
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	Antimony	0.14	mg/kg	0.14	mg/kg	M	4.3E-06	mg/kg-day	--	(mg/kg-day)-1	--
	Arsenic	1	mg/kg	1	mg/kg	M	3.1E-05	mg/kg-day	1.5E+00	(mg/kg-day)-1	4.6E-05
	Cadmium	0.08	mg/kg	0.08	mg/kg	M	2.4E-06	mg/kg-day	--	(mg/kg-day)-1	--
	Selenium	1.3	mg/kg	1.3	mg/kg	M	4.0E-05	mg/kg-day	--	(mg/kg-day)-1	--
	Silver	0.55	mg/kg	0.55	mg/kg	M	1.7E-05	mg/kg-day	--	(mg/kg-day)-1	--
	Zinc	49.7	mg/kg	49.7	mg/kg	M	1.5E-03	mg/kg-day	--	(mg/kg-day)-1	--
	4,4'-DDD	110	ug/kg	110	ug/kg	M	3.4E-06	mg/kg-day	2.4E-01	(mg/kg-day)-1	8.1E-07
	4,4'-DDE	120	ug/kg	120	ug/kg	M	3.7E-06	mg/kg-day	3.4E-01	(mg/kg-day)-1	1.2E-06
	bis(2-Ethylhexyl) Phthalate	320	ug/kg	320	ug/kg	M	9.8E-06	mg/kg-day	1.4E-02	(mg/kg-day)-1	1.4E-07
	Dieldrin	9.7	ug/kg	9.7	ug/kg	M	3.0E-07	mg/kg-day	1.6E+01	(mg/kg-day)-1	4.7E-06
	Heptachlor Epoxide	21	ug/kg	21	ug/kg	M	6.4E-07	mg/kg-day	9.1E+00	(mg/kg-day)-1	5.8E-06
										Total Risk	5.9E-05

(1) Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

-- - Cancer Slope Factor not available, therefore Cancer Risk not calculated.
N/A - Not Applicable.

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10/31/00

TABLE 9 RME
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
HORSESHOE ROAD COMPLEX SITE, SAYREVILLE, NEW JERSEY

Scenario Timeframe: Current and Future
Receptor Population: Residents
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Surface Water	Shellfish	AOC 6 - RR	Antimony	--	--	--	--	Antimony	Whole Body/ Blood	3.1E-02	--	--	3.1E-02
			Arsenic	4.6E-05	--	--	4.6E-05	Arsenic	Skin	3.0E-01	--	--	3.0E-01
			Cadmium	--	--	--	--	Cadmium	Kidney	7.1E-03	--	--	7.1E-03
			Selenium	--	--	--	--	Selenium	Liver	2.3E-02	--	--	2.3E-02
			Silver	--	--	--	--	Silver	Skin	9.8E-03	--	--	9.8E-03
			Zinc	--	--	--	--	Zinc	Blood	1.5E-02	--	--	1.5E-02
			4,4'-DDD	8.1E-07	--	--	8.1E-07	4,4'-DDD	--	--	--	--	--
			4,4'-DDE	1.2E-06	--	--	1.2E-06	4,4'-DDE	--	--	--	--	--
			bis(2-Ethylhexyl) Phthalate	1.4E-07	--	--	1.4E-07	bis(2-Ethylhexyl) Phthalate	Liver/Kidney	1.4E-03	--	--	1.4E-03
			Dieldrin	4.7E-06	--	--	4.7E-06	Dieldrin	Liver	1.7E-02	--	--	1.7E-02
			Heptachlor Epoxide	5.8E-06	--	--	5.8E-06	Heptachlor Epoxide	Liver	1.4E-01	--	--	1.4E-01
			(Total)	5.9E-05	--	--	5.9E-05	(Total)		5.5E-01	--	--	5.5E-01
Surface Water	Surface Water	AOC 6 - RR	Aluminum	--	--	--	--	Aluminum	--	2.8E-04	--	1.3E-04	4.1E-04
			Antimony	--	--	--	--	Antimony	Whole Body/ Blood	1.7E-03	--	8.0E-04	2.5E-03
			Arsenic	1.2E-06	--	5.7E-07	1.8E-06	Arsenic	Skin	8.0E-03	--	3.7E-03	1.2E-02
			Copper	--	--	--	--	Copper	--	7.5E-04	--	3.5E-04	1.1E-03
			Manganese	--	--	--	--	Manganese	--	5.1E-04	--	2.4E-04	7.5E-04
			Thallium	--	--	--	--	Thallium	Liver/ Blood	8.6E-03	--	4.0E-03	1.3E-02
			Vanadium	--	--	--	--	Vanadium	None	3.2E-04	--	1.5E-04	4.7E-04
			(Total)	1.2E-06	--	5.7E-07	1.8E-06	(Total)		2.0E-02	--	9.4E-03	3.0E-02
Sediment	Sediment	AOC 6 - RR	Arsenic	1.1E-04	--	8.0E-05	1.9E-04	Arsenic	Skin	8.9E-01	--	5.3E-01	1.2E+00
			Copper	--	--	--	--	Copper	--	8.4E-03	--	2.1E-03	1.1E-02
			(Total)	1.1E-04	--	8.0E-05	1.9E-04	(Total)		7.0E-01	--	5.3E-01	1.2E+00
			Total Risk Across All Media and Exposure Routes				2.5E-04	Total Hazard Index Across All Media and All Exposure Routes					1.8E+00

Total (Skin) HI =	1.5E+00
Total (Liver) HI =	2.0E-01
Total (Whole body) HI =	3.4E-02
Total (Kidney) HI =	7.1E-03

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APPENDIX B
SUMMARY OF CONTAMINANTS IN ENVIRONMENTAL MEDIA

Horseshoe Road Complex
Sayreville, New Jersey
Crab Muscle

Cas Rn	Chemical Name	Analytic Method	Unit \\	Sample Code / Sample Name / Sample Date	RCM01 9/30/99		RCM04 9/30/99		RCM05 9/30/99		RCM07 9/30/99		RCM10 9/24/99		RCM11 9/24/99	
(Group Code)	**(Group Description)**															
bio-voa-s	**Created by SUPER on 06/09/2000**															
74-87-3	CHLOROMETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
74-83-9	BROMOMETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
75-01-4	VINYL CHLORIDE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
75-00-3	CHLOROETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
75-09-2	DICHLOROMETHANE	TCL-VOC	ug/kg		2	J	2	J	10	U	2	J	10	U	10	U
67-64-1	ACETONE	TCL-VOC	ug/kg		10	U	140		80		83	J	67		13	
75-15-0	CARBON DISULFIDE	TCL-VOC	ug/kg		11		8	J	10	U	8	J	10		10	U
75-35-4	1,1-DICHLOROETHYLENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
75-34-3	1,1-DICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
540-59-0	1,2-DICHLOROETHENE(TOTAL)	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
67-66-3	CHLOROFORM	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
107-06-2	1,2-DICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
78-93-3	2-BUTANONE	TCL-VOC	ug/kg		6	J	10	U	10	U	13	J	2	J	10	U
71-55-6	1,1,1-TRICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
56-23-5	CARBON TETRACHLORIDE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
75-27-4	BROMODICHLOROMETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
78-87-5	1,2-DICHLOROPROPANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
10061-01-5	cis-1,3-DICHLOROPROPENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
79-01-6	TRICHLOROETHYLENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
124-48-1	CHLORODIBROMOMETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
79-00-5	1,1,2-TRICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
71-43-2	BENZENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
10061-02-6	trans-1,3-DICHLOROPROPENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
75-25-2	TRIBOMOMETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
108-10-1	4-METHYL-2-PENTANONE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
591-78-6	METHYL N-BUTYL KETONE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
127-18-4	TETRACHLOROETHENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
79-34-5	1,1,2,2-TETRACHLOROETHANE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
108-88-3	METHYLBENZENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
108-90-7	CHLOROBENZENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
100-41-4	ETHYLBENZENE	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
100-42-5	STYRENE (MONOMER)	TCL-VOC	ug/kg		10	U	10	U	10	U	10	UJ	10	U	10	U
1330-20-7	XYLENES, TOTAL	TCL-VOC	ug/kg		10	U	10	U	2	J	10	UJ	10	U	10	U

Cas Rn	Chemical Name	Analytic Method	Unit \\	Sample Code Sample Name Sample Date	RCM13 9/24/99		RCM14 9/24/99		RCM15 9/24/99		RCM19 9/23/99		RCM23 9/30/99		RCMCOMP1 9/24/99	
(Group Code)	**(Group Description)**															
bio-voa-s	**Created by SUPER on 06/09/2000**															
74-87-3	CHLOROMETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
74-83-9	BROMOMETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
75-01-4	VINYL CHLORIDE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
75-00-3	CHLOROETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
75-09-2	DICHLOROMETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	2	J	10	U
67-64-1	ACETONE	TCL-VOC	ug/kg		10	U	61	J	56		140		140		88	
75-15-0	CARBON DISULFIDE	TCL-VOC	ug/kg		15		10	UJ	7	J	10		7	J	3	J
75-35-4	1,1-DICHLOROETHYLENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
75-34-3	1,1-DICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
540-59-0	1,2-DICHLOROETHENE(TOTAL)	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
67-66-3	CHLOROFORM	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
107-06-2	1,2-DICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
78-93-3	2-BUTANONE	TCL-VOC	ug/kg		2	J	10	UJ	3	J	2	J	10	U	10	U
71-55-6	1,1,1-TRICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
56-23-5	CARBON TETRACHLORIDE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
75-27-4	BROMODICHLOROMETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
78-87-5	1,2-DICHLOROPROPANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
10061-01-5	cis-1,3-DICHLOROPROPENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
79-01-6	TRICHLOROETHYLENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
124-48-1	CHLORODIBROMOMETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
79-00-5	1,1,2-TRICHLOROETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
71-43-2	BENZENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
10061-02-6	trans-1,3-DICHLOROPROPENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
75-25-2	TRIBOMOMETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
108-10-1	4-METHYL-2-PENTANONE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
591-78-6	METHYL N-BUTYL KETONE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
127-18-4	TETRACHLOROETHENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
79-34-5	1,1,2,2-TETRACHLOROETHANE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
108-88-3	METHYLBENZENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
108-90-7	CHLOROBENZENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
100-41-4	ETHYLBENZENE	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
100-42-5	STYRENE (MONOMER)	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U
1330-20-7	XYLENES, TOTAL	TCL-VOC	ug/kg		10	U	10	UJ	10	U	10	U	10	U	10	U

Cas Rn	Chemical Name	Analytic Method	Unit \\	Sample Code	RCM01		RCM04		RCM05		RCM07		RCM10		RCM11	
				Sample Name												
				Sample Date	9/30/99		9/30/99		9/30/99		9/30/99		9/24/99		9/24/99	
(Group Code)	(Group Description)															
bio-svoc-s	Created by SUPER on 06/09/2000															
108-95-2	PHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
111-44-4	bis(2-CHLOROETHYL) ETHER	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
95-57-8	2-CHLOROPHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
541-73-1	M-DICHLOROBENZENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
106-46-7	1,4-DICHLOROBENZENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
95-50-1	1,2-DICHLOROBENZENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
95-48-7	2-METHYLPHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	34	J	330	U
108-60-1	2,2'-OXYBIS(1-CHLOROPROPAN	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
106-44-5	4-METHYLPHENOL (p-CRESOL)	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
621-64-7	N-NITROSODI-n-PROPYLAMINE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
67-72-1	HEXACHLOROETHANE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
98-95-3	NITROBENZENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
78-59-1	3,5,5-TRIMETHYL-2-CYCLOHEXE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
88-75-5	2-NITROPHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
105-67-9	2,4-DIMETHYLPHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
111-91-1	bis(2-CHLOROETHOXY) METHAN	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
120-83-2	2,4-DICHLOROPHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
120-82-1	1,2,4-TRICHLOROBENZENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
91-20-3	NAPHTHALENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
106-47-8	P-CHLOROANILINE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
87-68-3	HEXACHLORO-1,3-BUTADIENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
59-50-7	4-CHLORO-3-METHYLPHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
91-57-6	2-METHYLNAPHTHALENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
77-47-4	HEXACHLOROCYCLOPENTADIE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
88-06-2	2,4,6-TRICHLOROPHENOL	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
95-95-4	2,4,5-TRICHLOROPHENOL	TCL-SVOC	ug/kg		800	U	800	U	800	U	800	U	800	U	800	U
91-58-7	2-CHLORONAPHTHALENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
88-74-4	2-NITROANILINE	TCL-SVOC	ug/kg		800	U	800	U	800	U	800	U	800	U	800	U
131-11-3	DIMETHYL PHTHALATE	TCL-SVOC	ug/kg		330	U	330	U	330	U	35	J	330	U	330	U
208-96-8	ACENAPHTHYLENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
606-20-2	2,6-DINITROTOLUENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U
99-09-2	3-NITROANILINE	TCL-SVOC	ug/kg		800	U	800	U	800	U	800	U	800	U	800	U
83-32-9	ACENAPHTHENE	TCL-SVOC	ug/kg		330	U	330	U	330	U	330	U	330	U	330	U

Cas Rn	Chemical Name	Analytic Method	Sample Code / Sample Name / Sample Date / Unit \\	RCM13 9/24/99	RCM14 9/24/99	RCM15 9/24/99	RCM19 9/23/99	RCM23 9/30/99	RCMCOMP1 9/24/99
(Group Code)	**(Group Description)**								
bio-svoc-s	**Created by SUPER on 06/09/2000**								
108-95-2	PHENOL	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
111-44-4	bis(2-CHLOROETHYL) ETHER	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
95-57-8	2-CHLOROPHENOL	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
541-73-1	M-DICHLOROBENZENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
106-46-7	1,4-DICHLOROBENZENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
95-50-1	1,2-DICHLOROBENZENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
95-48-7	2-METHYLPHENOL	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
108-60-1	2,2'-OXYBIS(1-CHLOROPROPAN	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
106-44-5	4-METHYLPHENOL (p-CRESOL)	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
621-64-7	N-NITROSODI-n-PROPYLAMINE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
67-72-1	HEXACHLOROETHANE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
98-95-3	NITROBENZENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
78-59-1	3,5,5-TRIMETHYL-2-CYCLOHEXE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
88-75-5	2-NITROPHENOL	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
105-67-9	2,4-DIMETHYLPHENOL	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
111-91-1	bis(2-CHLOROETHOXY) METHAN	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
120-83-2	2,4-DICHLOROPHENOL	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
120-82-1	1,2,4-TRICHLOROBENZENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
91-20-3	NAPHTHALENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
106-47-8	P-CHLOROANILINE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
87-68-3	HEXACHLORO-1,3-BUTADIENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
59-50-7	4-CHLORO-3-METHYLPHENOL	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
91-57-6	2-METHYLNAPHTHALENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
77-47-4	HEXACHLOROCYCLOPENTADIE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
88-06-2	2,4,6-TRICHLOROPHENOL	TCL-SVOC	ug/kg	330 U	330 U	800 U	800 U	330 U	330 U
95-95-4	2,4,5-TRICHLOROPHENOL	TCL-SVOC	ug/kg	800 U	800 U	330 U	330 U	800 U	800 U
91-58-7	2-CHLORONAPHTHALENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
88-74-4	2-NITROANILINE	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
131-11-3	DIMETHYL PHTHALATE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
208-96-8	ACENAPHTHYLENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
606-20-2	2,6-DINITROTOLUENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
99-09-2	3-NITROANILINE	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
83-32-9	ACENAPHTHENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U

Cas Rn	Chemical Name	Analytic Method	Sample Code / Sample Name / Sample Date / Unit \\	RCM01 9/30/99	RCM04 9/30/99	RCM05 9/30/99	RCM07 9/30/99	RCM10 9/24/99	RCM11 9/24/99
51-28-5	2,4-DINITROPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
100-02-7	4-NITROPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
132-64-9	DIBENZOFURAN	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
121-14-2	2,4-DINITROTOLUENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
84-66-2	DIETHYL PHTHALATE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
7005-72-3	4-CHLOROPHENYL PHENYL ETH	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
86-73-7	FLUORENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
100-01-6	P-NITROANILINE	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
534-52-1	4,6-DINITRO-2-METHYLPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
86-30-6	N-NITROSODIPHENYLAMINE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
101-55-3	4-BROMOPHENYL PHENYL ETHE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
118-74-1	HEXACHLOROBENZENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
87-86-5	PENTACHLOROPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
85-01-8	PHENANTHRENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
120-12-7	ANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
86-74-8	CARBAZOLE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
84-74-2	DI-n-BUTYL PHTHALATE	TCL-SVOC	ug/kg	270 U	270 U	72 J	95 J	390	330
206-44-0	FLUORANTHENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
129-00-0	PYRENE	TCL-SVOC	ug/kg	330 U	51 J	330 U	330 U	330 U	330 U
85-68-7	BENZYL BUTYL PHTHALATE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
91-94-1	3,3'-DICHLOROBENZIDINE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
56-55-3	BENZO(a)ANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
218-01-9	1,2-BENZPHENANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
117-81-7	bis(2-ETHYLHEXYL) PHTHALATE	TCL-SVOC	ug/kg	66 J	52 J	52 J	71 J	320 J	220 J
117-84-0	DI-N-OCTYL PHTHALATE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
205-99-2	BENZO(b)FLUORANTHENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
207-08-9	BENZO(k)FLUORANTHENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
50-32-8	BENZO(a)PYRENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
193-39-5	INDENO(1,2,3-c,d)PYRENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
53-70-3	DIBENZ(a,h)ANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
191-24-2	BENZO(g,h,i)PERYLENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U

Cas Rn	Chemical Name	Analytic Method	Sample Code / Sample Name / Sample Date / Unit \\	RCM13 9/24/99	RCM14 9/24/99	RCM15 9/24/99	RCM19 9/23/99	RCM23 9/30/99	RCMCOMP1 9/24/99
51-28-5	2,4-DINITROPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
100-02-7	4-NITROPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
132-64-9	DIBENZOFURAN	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
121-14-2	2,4-DINITROTOLUENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
84-66-2	DIETHYL PHTHALATE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
7005-72-3	4-CHLOROPHENYL PHENYL ETH	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
86-73-7	FLUORENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
100-01-6	P-NITROANILINE	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
534-52-1	4,6-DINITRO-2-METHYLPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
86-30-6	N-NITROSODIPHENYLAMINE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
101-55-3	4-BROMOPHENYL PHENYL ETHE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
118-74-1	HEXACHLOROBENZENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
87-86-5	PENTACHLOROPHENOL	TCL-SVOC	ug/kg	800 U	800 U	800 U	800 U	800 U	800 U
85-01-8	PHENANTHRENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
120-12-7	ANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
86-74-8	CARBAZOLE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
84-74-2	DI-n-BUTYL PHTHALATE	TCL-SVOC	ug/kg	150 J	180 J	330 U	180 J	330 U	150 J
206-44-0	FLUORANTHENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
129-00-0	PYRENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
85-68-7	BENZYL BUTYL PHTHALATE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
91-94-1	3,3'-DICHLOROBENZIDINE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
56-55-3	BENZO(a)ANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
218-01-9	1,2-BENZPHENANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	53 J	330 U
117-81-7	bis(2-ETHYLHEXYL) PHTHALATE	TCL-SVOC	ug/kg	200 J	250 J	280 J	200 J	330 U	160 J
117-84-0	DI-N-OCTYL PHTHALATE	TCL-SVOC	ug/kg	330 U	330 U	330 U	330 U	330 U	330 U
205-99-2	BENZO(b)FLUORANTHENE	TCL-SVOC	ug/kg	330 U	330 U		330 U	330 U	330 U
207-08-9	BENZO(k)FLUORANTHENE	TCL-SVOC	ug/kg	330 U	330 U		330 U	330 U	330 U
50-32-8	BENZO(a)PYRENE	TCL-SVOC	ug/kg	330 U	330 U		330 U	330 U	330 U
193-39-5	INDENO(1,2,3-c,d)PYRENE	TCL-SVOC	ug/kg	330 U	330 U		330 U	330 U	330 U
53-70-3	DIBENZ(a,h)ANTHRACENE	TCL-SVOC	ug/kg	330 U	330 U		330 U	330 U	330 U
191-24-2	BENZO(g,h,i)PERYLENE	TCL-SVOC	ug/kg	330 U	330 U		330 U	330 U	330 U

Cas Rn	Chemical Name	Analytic Method	Unit \\	Sample Code / Sample Name / Sample Date	RCM01 9/30/99		RCM04 9/30/99		RCM05 9/30/99		RCM07 9/30/99		RCM10 9/24/99		RCM11 9/24/99	
(Group Code)	**(Group Description)**															
bio-pest-s	**Created by SUPER on 06/09/2000**															
319-84-6	ALPHA BHC	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
319-85-7	BETA BHC	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
319-86-8	DELTA BHC	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
58-89-9	GAMMA-BHC (LINDANE)	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
76-44-8	HEPTACHLOR	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
309-00-2	ALDRIN	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
1024-57-3	HEPTACHLOR EPOXIDE	TCL-P/PCB	ug/kg		5	U	5	U	21	P	8.7	J	9.4	J	5	U
959-98-8	ENDOSULFAN I	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
60-57-1	DIELDRIN	TCL-P/PCB	ug/kg		5	U	5	U	9.7		5	U	5	U	5	U
72-55-9	4,4'-DDE	TCL-P/PCB	ug/kg		5	U	5	U	120		5	U	28	J	8.4	J
72-20-8	ENDRIN	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
33213-65-9	ENDOSULFAN II	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	4.5	J
72-54-8	4,4'-DDD	TCL-P/PCB	ug/kg		5	U	5	U	110		5	U	5	U	5	U
1031-07-8	ENDOSULFAN SULFATE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
50-29-3	4,4'-DDT	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
72-43-5	1,1,1-TRICHLORO-2,2-BIS (P-MET	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
53494-70-5	ENDRIN KETONE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
7421-93-4	ENDRIN ALDEHYDE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
5103-71-9	ALPHA-CHLORDANE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
5103-74-2	GAMMA-CHLORDANE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U	5	U
8001-35-2	CAMPHECHLOR	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U	100	U
12674-11-2	AROCLOR-1016	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U	100	U
11104-28-2	AROCLOR-1221	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U	100	U
11141-16-5	AROCLOR-1232	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U	100	U
53469-21-9	AROCLOR-1242	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U	100	U
12672-29-6	AROCLOR-1248	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U	100	U
11097-69-1	AROCLOR-1254	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U	100	U
11096-82-5	AROCLOR-1260	TCL-P/PCB	ug/kg		100	U	400	U	400	U	100	U	100	U	100	U

Cas Rn	Chemical Name	Analytic Method	Unit \\	Sample Code / Sample Name / Sample Date	RCM13 9/24/99		RCM14 9/24/99		RCM15 9/24/99		RCM19 9/23/99		RCM23 9/30/99		RCMCOMP1 9/24/99	
(Group Code)	(Group Description)															
bio-pest-s	Created by SUPER on 06/09/2000															
319-84-6	ALPHA BHC	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
319-85-7	BETA BHC	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
319-86-8	DELTA BHC	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
58-89-9	GAMMA-BHC (LINDANE)	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
76-44-8	HEPTACHLOR	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
309-00-2	ALDRIN	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
1024-57-3	HEPTACHLOR EPOXIDE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
959-98-8	ENDOSULFAN I	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
60-57-1	DIELDRIN	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
72-55-9	4,4'-DDE	TCL-P/PCB	ug/kg		12	J	5	U	11	J	5	U	5	U		R
72-20-8	ENDRIN	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
33213-65-9	ENDOSULFAN II	TCL-P/PCB	ug/kg		5	U	11	J	5	U	5	U	5	U		R
72-54-8	4,4'-DDD	TCL-P/PCB	ug/kg		11	JN	18	J	5	U	5.3	J	5	U		R
1031-07-8	ENDOSULFAN SULFATE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
50-29-3	4,4'-DDT	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
72-43-5	1,1,1-TRICHLORO-2,2-BIS (P-MET	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
53494-70-5	ENDRIN KETONE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
7421-93-4	ENDRIN ALDEHYDE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
5103-71-9	ALPHA-CHLORDANE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
5103-74-2	GAMMA-CHLORDANE	TCL-P/PCB	ug/kg		5	U	5	U	5	U	5	U	5	U		R
8001-35-2	CAMPHECHLOR	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U		R
12674-11-2	AROCLOR-1016	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U		R
11104-28-2	AROCLOR-1221	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U		R
11141-16-5	AROCLOR-1232	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U		R
53469-21-9	AROCLOR-1242	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U		R
12672-29-6	AROCLOR-1248	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U		R
11097-69-1	AROCLOR-1254	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	100	U		R
11096-82-5	AROCLOR-1260	TCL-P/PCB	ug/kg		100	U	100	U	100	U	100	U	400	U		R

Cas Rn	Chemical Name	Analytic Method	Unit \\	Sample Code		RCM01		RCM04		RCM05		RCM07		RCM10		RCM11	
				Sample Name													
				Sample Date		9/30/99		9/30/99		9/30/99		9/30/99		9/24/99		9/24/99	
(Group Code)	**(Group Description)**																
4-MET-S	**Created by SUPER on 06/08/2000**																
7429-90-5	ALUMINUM (F	TAL-METALS	mg/kg				R	6	B		R		R	3.8		4.9	
7440-36-0	ANTIMONY	TAL-METALS	mg/kg			0.09	B	0.07	U	0.13	BJ	0.07	UJ		R	0.08	BJ
7440-38-2	ARSENIC	TAL-METALS	mg/kg			0.83	J	0.63		0.63		0.67	J	0.79	J	0.69	
7440-39-3	BARIUM	TAL-METALS	mg/kg			0.28	B	0.11	B	0.23	B	0.22	B	0.19	B	0.58	B
7440-41-7	BERYLLIUM	TAL-METALS	mg/kg			0.02	U	0.02	U	0.04	B	0.02	UJ	0.02	U	0.02	U
7440-43-9	CADMIUM	TAL-METALS	mg/kg			0.07	B	0.02	U	0.02	UJ	0.05	BJ	0.02	U	0.02	U
7440-70-2	CALCIUM MET	TAL-METALS	mg/kg			1320	J	1040	J	1360	J	1270	E	1370	J	2700	J
7440-47-3	CHROMIUM	TAL-METALS	mg/kg			0.12	B	0.13	B	0.08	B	0.1	B	0.14	B	0.19	B
7440-48-4	COBALT	TAL-METALS	mg/kg			0.03	U	0.03	U	0.03	U	0.03	U	0.03	U	0.03	U
7440-50-8	COPPER	TAL-METALS	mg/kg			17.3		11.4		13		13.6			R		R
7439-89-6	IRON	TAL-METALS	mg/kg			8.1	J	3.7	B	18.1	J	10.8	J	2.7	BE*		R
7439-92-1	LEAD	TAL-METALS	mg/kg			0.49	J	0.46	J	0.44	J	0.42		1.3	*	0.91	U*
7439-95-4	MAGNESIUM	TAL-METALS	mg/kg			403	J	279	J	319	J	402	J	398	J	424	J
7439-96-5	MANGANESE	TAL-METALS	mg/kg				R		R		R		R	0.71	J	2.3	J
7439-97-6	MERCURY	TAL-METALS	mg/kg			0.03	UM*	0.05	M	0.03	UM	0.03	UM	0.04		0.03	U
7440-02-0	NICKEL	TAL-METALS	mg/kg			0.04	UJ	0.04	UJ	0.04	UJ	0.04	UJ	0.51	BJ	0.23	BJ
7440-09-7	POTASSIUM	TAL-METALS	mg/kg			2270		1860		1710		1970		1860	J	2450	J
7782-49-2	SELENIUM	TAL-METALS	mg/kg			0.7	NJ	0.4	NJ	0.44	NJ	0.5	NJ	0.76	J	0.47	J*
7440-22-4	SILVER	TAL-METALS	mg/kg			0.06	BNJ	0.02	UNJ	0.02	UNJ	0.05	BNJ	0.21	B	0.28	B
7440-23-5	SODIUM	TAL-METALS	mg/kg			5680		4570		5590		4730		3840	J	2860	J
7440-28-0	THALLIUM	TAL-METALS	mg/kg			0.13	UNJ	0.14	UNJ	0.13	UNJ	0.13	UNJ		R		R
7440-62-2	VANADIUM (F	TAL-METALS	mg/kg			0.03	U	0.03	U	0.03	U	0.03	U	0.03	U	0.03	U
7440-66-6	ZINC	TAL-METALS	mg/kg			45.2	NJ	39.7	NJ	36.6	NJ	33.4	NJ	47.7	J	49.7	J
57-12-5	CYANIDE	TAL-METALS	mg/kg														

Cas Rn	Chemical Name	Analytic Method	Unit \\	Sample Code	RCM13		RCM14		RCM15		RCM19		RCM23		RCMCOMP1	
				Sample Name												
				Sample Date	9/24/99		9/24/99		9/24/99		9/23/99		9/30/99		9/24/99	
(Group Code)	(Group Description)															
4-MET-S	Created by SUPER on 06/08/2000															
7429-90-5	ALUMINUM (F	TAL-METALS	mg/kg		4.2		3.7		5.7		3.5		5.8	B	3.7	
7440-36-0	ANTIMONY	TAL-METALS	mg/kg		0.07	UJ	0.14	BJ		R	0.12	BJ	0.09	B	0.07	UJ
7440-38-2	ARSENIC	TAL-METALS	mg/kg		0.81		0.86		1	J	0.6		0.48		0.67	
7440-39-3	BARIUM	TAL-METALS	mg/kg		0.19	B	0.23	B	0.13		0.31	B	0.18	B	0.2	
7440-41-7	BERYLLIUM	TAL-METALS	mg/kg		0.02	U	0.02	U	0.02		0.02	U	0.03	B	0.02	U
7440-43-9	CADMIUM	TAL-METALS	mg/kg		0.03	B	0.08	B	0.04	B	0.05	B	0.02	U	0.07	B
7440-70-2	CALCIUM MET	TAL-METALS	mg/kg		453	J	686	J	749	J	833	J	1070	J	579	J
7440-47-3	CHROMIUM	TAL-METALS	mg/kg		0.12	B	0.11	B	0.15	B	0.11	B	0.1	B	0.09	B
7440-48-4	COBALT	TAL-METALS	mg/kg		0.03	U	0.03	U	0.03	U	0.03	U	0.03	U	0.03	U
7440-50-8	COPPER	TAL-METALS	mg/kg			R		R		R		R	12.4			R
7439-89-6	IRON	TAL-METALS	mg/kg			R		R		R		R	11.6	J		R
7439-92-1	LEAD	TAL-METALS	mg/kg			R		R	0.86	U	1.3	*	0.46	J	0.92	U*
7439-95-4	MAGNESIUM	TAL-METALS	mg/kg		291	J	308	J	342	J	310	J	361	EJ	292	J
7439-96-5	MANGANESE	TAL-METALS	mg/kg		0.65	BJ	1.1	J	0.58	BE*	1.1	J		R	0.47	BE*
7439-97-6	MERCURY	TAL-METALS	mg/kg		0.03	U	0.03	U	0.03	U	0.03	U	0.03	UM	0.07	M
7440-02-0	NICKEL	TAL-METALS	mg/kg		0.07	BJ	0.11	BJ	0.08	BJ	0.05	BJ	0.04	UJ	0.06	BJ
7440-09-7	POTASSIUM	TAL-METALS	mg/kg		2620	J	2580	J	2610	J	2510	J	2100		2130	J
7782-49-2	SELENIUM	TAL-METALS	mg/kg		0.5	J*	0.81	J	1.3	*J	0.76	J	0.65	NJ	0.61	*J
7440-22-4	SILVER	TAL-METALS	mg/kg		0.31	B	0.28	B	0.55		0.23	B	0.02	UNJ	0.74	
7440-23-5	SODIUM	TAL-METALS	mg/kg		2840	J	2680	J	3490	J	2750	J	4580		3500	J
7440-28-0	THALLIUM	TAL-METALS	mg/kg			R		R		R		R	0.12	UNJ		R
7440-62-2	VANADIUM (F	TAL-METALS	mg/kg		0.03	U	0.03	U	0.02	U	0.02	U	0.03	U	0.03	U
7440-66-6	ZINC	TAL-METALS	mg/kg		26.7	J	28.2	J	39.5	J	25.7	J	45.1	NJ	20.4	J
57-12-5	CYANIDE	TAL-METALS	mg/kg													

APPENDIX C
CONCENTRATION-TOXICITY SCREENS

APPENDIX C

CHEMICAL CONCENTRATION - TOXICITY SCREEN
SHELLFISH MUSCLE - RARITAN RIVER (RR)
HORSESHOE ROAD COMPLEX SITE, SAYREVILLE, NEW JERSEY

CARCINOGENS:

CHEMICAL	Chemical of Potential Concern (Contributes >1%)	Maximum Detected Concentration (mg/kg)	Slope Factor (mg/kg-day)-1	Risk Factor	Contribution to Total Risk for Matrix (Percent)
Arsenic	YES	1.00E+00	1.5E+00	1.50E+00	78.19%
1,2-Benzphenanthracene (Chrysene)	no	5.30E-02	7.3E-03	3.87E-04	0.02%
4,4'-DDD	YES	1.10E-01	2.4E-01	2.64E-02	1.38%
4,4'-DDE	YES	1.20E-01	3.4E-01	4.08E-02	2.13%
Bis(2-ethylhexyl)phthalate	no	3.20E-01	1.4E-02	4.48E-03	0.23%
Dieldrin	YES	9.70E-03	1.6E+01	1.55E-01	8.09%
Heptachlor Epoxide	YES	2.10E-02	9.1E+00	1.91E-01	9.96%
Methylene Chloride	no	2.00E-03	7.5E-03	1.50E-05	0.00%
			TOTAL RISK FACTOR	1.92E+00	100%

NONCARCINOGENS:

CHEMICAL	Chemical of Potential Concern (Contributes >1%)	Maximum Detected Concentration (mg/kg)	Reference Dose (mg/kg-day)	Risk Factor	Contribution to Total Risk for Matrix (Percent)
Aluminum	no	6.00E+00	1.0E+00	6.00E+00	0.10%
Antimony	YES	1.40E-01	4.0E-04	3.50E+02	5.59%
Arsenic	YES	1.00E+00	3.0E-04	3.33E+03	53.26%
Barium	no	5.80E-01	7.0E-02	8.29E+00	0.13%
Beryllium	no	4.00E-02	2.0E-03	2.00E+01	0.32%
Cadmium (food)	YES	8.00E-02	1.0E-03	8.00E+01	1.28%
Chromium III (insoluble salts)	no	1.58E-01	1.5E+00	1.05E-01	0.00%
Chromium VI	no	3.23E-02	3.0E-03	1.08E+01	0.17%
Manganese	no	2.30E+00	1.4E-01	1.64E+01	0.26%
Nickel (soluble salt)	no	5.10E-01	2.0E-02	2.55E+01	0.41%
Selenium	YES	1.30E+00	5.0E-03	2.60E+02	4.15%
Silver	YES	7.40E-01	5.0E-03	1.48E+02	2.36%
Zinc (and compounds)	YES	4.97E+01	3.0E-01	1.66E+02	2.65%
2-Butanone	no	1.30E-02	6.0E-01	2.17E-02	0.00%
2-Methylphenol	no	3.40E-02	5.0E-02	6.80E-01	0.01%
Acetone	no	1.40E-01	1.0E-01	1.40E+00	0.02%
Bis(2-ethylhexyl)phthalate	no	3.20E-01	2.0E-02	1.60E+01	0.26%
Carbon Disulfide	no	1.50E-02	1.0E-01	1.50E-01	0.00%
Dieldrin	YES	9.70E-03	5.0E-05	1.94E+02	3.10%
Di-n-butyl phthalate	no	3.90E-01	1.0E-01	3.90E+00	0.06%
Endosulfan II	no	1.10E-02	6.0E-03	1.83E+00	0.03%
Heptachlor Epoxide	YES	2.10E-02	1.3E-05	1.62E+03	25.81%
Methylene Chloride	no	2.00E-03	6.0E-02	3.33E-02	0.00%
Pyrene	no	5.10E-02	3.0E-02	1.70E+00	0.03%
Xylenes (Total)	no	2.00E-03	2.0E+00	1.00E-03	0.00%
			TOTAL RISK FACTOR	6.26E+03	100%

400374

10/31/00

APPENDIX D
CHEMICAL CONTAMINANTS OF CONCERN

APPENDIX D

CHEMICAL CONTAMINANTS OF CONCERN
HORSESHOE ROAD COMPLEX, SAYREVILLE, NEW JERSEY

Media	Exposure Media	Area of Concern
Surface Water	Shellfish	AOC-6/RR
		Antimony
		Arsenic
		Cadmium
		Selenium
		Silver
		Zinc
		4,4'-DDD
		4,4'-DDE
		bis(2-Ethylhexyl) Phthalate
		Dieldrin
		Heptachlor Epoxide

400376

10/31/00

APPENDIX E
TOXICITY PROFILES

Mercury, elemental; CASRN 7439-97-6

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Mercury, elemental

File On-Line 09/07/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	on-line	06/01/1995
Carcinogenicity Assessment (II.)	on-line	05/01/1995

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6

Not available at this time.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 06/01/1995

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
Hand tremor; increases in memory disturbances; slight subjective and objective evidence of autonomic dysfunction	NOAEL: None LOAEL: 0.025 mg/cu.m (converted to LOAEL [ADJ] of 0.009 mg/cu.m	30	1	3E-4 mg/cu.m

Human occupational
inhalation studies

Fawer et al., 1983;
Piikivi and Tolonen, 1989;
Piikivi and Hanninen, 1989;
Piikivi, 1989;
Ngim et al., 1992;
Liang et al., 1993

*Conversion Factors and Assumptions: This is an extrarespiratory effect of a vapor (gas). The LOAEL is based on an 8-hour TWA occupational exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. LOAEL(HEC) =LOAEL(ADJ) = 0.025 mg/cu.m x MVho/MVh x 5 days/7 days = 0.009 mg/cu.m. Air concentrations (TWA) were measured in the Fawer et al. (1983), Ngim et al. (1992), and Liang et al. (1993) studies. Air concentrations were extrapolated from blood levels based on the conversion factor of Roels et al. (1987) as described in the Additional Comments section for the studies of Piikivi and Tolonen (1989), Piikivi and Hanninen (1989), and Piikivi (1989).

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

Fawer, R.F., U. DeRibaupierre, M.P. Guillemin, M. Berode and M. Lobe. 1983. Measurement of hand tremor induced by industrial exposure to metallic mercury. J. Ind. Med. 40: 204-208.

Piikivi, L. and U. Tolonen. 1989. EEG findings in chlor-alkali workers subjected to low long term exposure to mercury vapor. Br. J. Ind. Med. 46: 370-375.

Piikivi, L. and H. Hanninen. 1989. Subjective symptoms and psychological performance of chlorine-alkali workers. Scand. J. Work Environ. Health. 15: 69-74.

Piikivi, L. 1989. Cardiovascular reflexes and low long-term exposure to mercury vapor. Int. Arch. Occup. Environ. Health. 61: 391-395.

Ngim, C.H., S.C. Foo, K.W. Boey and J. Jeyaratnam. 1992. Chronic neurobehavioral effects of elemental mercury in dentists. Br. J. Ind. Med. 49: 782-790.

Liang, Y-X., R-K. Sun, Y. Sun, Z-Q. Chen and L-H. Li. 1993. Psychological effects of low exposure to mercury vapor: Application of a computer-administered neurobehavioral evaluation system. Environ. Res. 60: 320-327.

Fawer et al. (1983) used a sensitive objective electronic measure of intention tremor (tremors that occur at the initiation of voluntary movements) in 26 male workers (mean age of 44 years) exposed to low levels of mercury vapor in various occupations: fluorescent tube manufacture (n=7), chloralkali plants (n=12), and acetaldehyde production (n=7). Controls (n=25; mean age of 44.6 years) came from the same factories but were not exposed occupationally. Personal air samples (two per subject) were used to characterize an average exposure concentration of 0.026 mg/cu.m. It should be noted that it is likely that the levels of mercury in the air varied during the period of exposure and historical data indicate that previous exposures may have been higher. Exposure measurements for the control cohort were not performed. The average duration of exposure was 15.3 years. The measures of tremor were significantly increased in the exposed compared to control cohorts, and were shown to correspond to exposure and not to chronologic age. These findings are consistent with neurophysiological impairments that might result from

accumulation of mercury in the cerebellum and basal ganglia. Thus, the TWA of 0.026 mg/cu.m was designated a LOAEL. Using the TWA and adjusting for occupational ventilation rates and workweek, the resultant LOAEL(HEC) is 0.009 mg/cu.m.

Piikivi and Tolonen (1989) used EEGs to study the effects of long-term exposure to mercury vapor in 41 chloralkali workers exposed for a mean of 15.6 +/- 8.9 years as compared with matched referent controls. They found that the exposed workers, who had mean blood Hg levels of 12 ug/L and mean urine Hg levels of 20 ug/L, tended to have an increased number of EEG abnormalities when analyzed by visual inspection only. When the EEGs were analyzed by computer, however, the exposed workers were found to have significantly slower and attenuated brain activity as compared with the referents. These changes were observed in 15% of the exposed workers. The frequency of these changes correlated with cortical Hg content (measured in other studies); the changes were most prominent in the occipital cortex less prominent in the parietal cortex, and almost absent in the frontal cortex. The authors extrapolated an exposure level associated with these EEG changes of 0.025 mg/cu.m from blood levels based on the conversion factor calculated by Roels et al. (1987).

Piikivi and Hanninen (1989) studied the subjective symptoms and psychological performances on a computer-administered test battery in 60 chloralkali workers exposed to mercury vapor for a mean of 13.7 +/- 5.5 years as compared with matched referent controls. The exposed workers had mean blood Hg levels of 10 ug/L and mean urine Hg levels of 17 ug/L. A statistically significant increase in subjective measures of memory disturbance and sleep disorders was found in the exposed workers. The exposed workers also reported more anger, fatigue and confusion. No objective disturbances in perceptual motor, memory or learning abilities were found in the exposed workers. The authors extrapolated an exposure level associated with these subjective measures of memory disturbance of 0.025 mg/cu.m from blood levels based on the conversion factor calculated by Roels et al. (1987).

Both subjective and objective symptoms of autonomic dysfunction were investigated in 41 chloralkali workers exposed to mercury vapor for a mean of 15.6 +/- 8.9 years as compared with matched referent controls (Piikivi, 1989). The quantitative non-invasive test battery consisted of measurements of pulse rate variation in normal and deep breathing, in the Valsalva maneuver and in vertical tilt, as well as blood pressure responses during standing and isometric work. The exposed workers had mean blood levels of 11.6 ug/L and mean urine levels of 19.3 ug/L. The exposed workers complained of more subjective symptoms of autonomic dysfunction than the controls, but the only statistically significant difference was an increased reporting of palpitations in the exposed workers. The quantitative tests revealed a slight decrease in pulse rate variations, indicative of autonomic reflex dysfunction, in the exposed workers. The authors extrapolated an exposure level associated with these subjective and objective measures of autonomic dysfunction of 0.030 mg/cu.m from blood levels based on the conversion factor calculated by Roels et al. (1987).

Two more recent studies in other working populations corroborate the neurobehavioral toxicity of low-level mercury exposures observed in the Fawer et al. (1983), Piikivi and Tolonen (1989), Piikivi and Hanninen (1989), and Piikivi (1989) studies.

Ngim et al. (1992) assessed neurobehavioral performance in a cross-sectional study of 98 dentists (38 female, 60 male; mean age 32, range 24-49 years) exposed to TWA concentrations of 0.014 mg/cu.m (range 0.0007 to 0.042 mg/cu.m) versus 54 controls (27 female, 27 male; mean age 34, range 23-50 years) with no history of occupational exposure to mercury. Air concentrations were measured with personal sampling badges over typical working hours (8-10 hours) and converted to an 8-hour TWA. No details on the number of exposure samples or exposure histories were provided. Blood samples from the exposed cohort were also taken and the data supported the correspondence calculated by Roels et al. (1987). Based on extrapolation of the average blood mercury concentration (9.8 ug/L), the average exposure concentration would be estimated at 0.023 mg/cu.m. The average duration of practice of the exposed dentists was 5.5 years. Exposure measurements of the control cohort were not performed. The exposed and control groups were adequately matched for age, amount of fish consumption, and number of amalgam dental fillings. The performance of the dentists was significantly worse than controls on a number of neurobehavioural tests measuring motor speed (finger tapping), visual scaning, visumotor coordination and concentration, visual memory, and visuomotor coordination speed. These neurobehavioral effects are consistent with central and peripheral neurotoxicity and the TWA is considered a LOAEL. Using the TWA and adjusting for occupational ventilation rates and the reported 6-day workweek, the resultant LOAEL(HEC) is 0.006 mg/cu.m.

Liang et al. (1993) investigated workers in a fluorescent lamp factory with a computer-adminstered neurobehavioral evaluation system and a mood inventory profile. The exposed cohort (mean age 34.2 years) consisted of 19 females and 69 males exposed to ninterruptedly for at least 2 years prior to the study. Exposure was monitored with area samplers and ranged from 0.008 to 0.085 mg/cu.m across worksites. No details on how the exposure profiles to account for time spent in different worksites were constructed. The average exposure was estimated at 0.033 mg/cu.m. (range 0.005 to 0.19 mg/cu.m). The average duration was of working was 15.8 years for the exposed cohort. Urinary excretion was also monitored and reported to average 0.025 mg/L. The control cohort (mean age 35.1 years) consisted of 24 females and 46 males recruited from an embroidery factory. The controls were matched for age, education, smoking and drinking habits. Exposure measurements for the control cohort were not performed. The exposed cohort performed significantly worse than the control on tests of finger tapping, mental arithmetic, two-digit searches, switiching attention, and visual reaction time. The effect on performance persisted after the confounding factor of chronological age was controlled. Based on these neurobehavioral effects, the TWA of 0.033 mg/cu.m is designated as LOAEL. Using the TWA and adjusting for occupational ventilation rates and workweek, the resultant LOAEL(HEC) is 0.012 mg/cu.m.

The above studies were taken together as evidence for a LOAEL based on neurobehavioral effects of low-level mercury exposures. The LOAEL(HEC) levels calculated on measured air concentration levels of the Ngim et al. (1992) and the Liang et al. (1993) studies bracket that calculated based on the air concentrations measured by Fawer et al. (1983) as a median HEC level. Extrapolations of blood levels, used as biological monitoring that accounts for variability in exposure levels, also converge at 0.025 mg/cu.m as a TWA which results in the same HEC level. Thus, the TWA level of 0.025 mg/cu.m was used to represent the exposure for the synthesis of the studies described above. Using this TWA and taking occupational ventilation rates and workweek into account results in a LOAEL(HEC) of 0.009 mg/cu.m.

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF -- An uncertainty factor of 10 was used for the protection of sensitive human subpopulations (including concern for acrodynia - see Additional Comments section) together with the use of a LOAEL. An uncertainty factor of 3 was used for lack of data base, particularly developmental and reproductive studies.

MF -- None

___I.B.4. ADDITIONAL COMMENTS (INHALATION RfC)

Probably the most widely recognized form of hypersensitivity to mercury poisoning is the uncommon syndrome known as acrodynia, also called erythredema polyneuropathy or pink disease (Warkany and Hubbard, 1953). Infantile acrodynia was first described in 1828, but adult cases have also since been reported. While acrodynia has generally been associated with short-term exposures and with urine levels of 50 ug/L or more, there are some cases in the literature in which mercury exposure was known to have occurred, but no significant (above background) levels in urine were reported. There could be many reasons for this, but the most likely is that urine levels are not a simple measure of body burden or of target tissue (i.e., brain levels); however, they are the best means available for assessing the extent of exposure. It was felt that the RfC level estimated for mercury vapor based on neurotoxicity of chronic exposure in workers is adequate to protect children from risk of acrodynia because such exposures of long duration would be expected to raise urine levels by only 0.12 ug/L against a background level of up to 20 ug/L (i.e., such exposures would not add significantly to the background level of mercury in those exposed).

Roels et al. (1987) investigated the relationships between the concentrations of metallic mercury in air and levels monitored in blood or urine in workers exposed during manufacturing of dry alkaline batteries. Breathing zone personal samples were used to characterize airborne mercury vapors. Total mercury in blood and urine samples were analyzed using atomic absorption. The investigation controlled for several key factors including the use of reliable personal air monitoring, quality control for blood and urine analyses, standardization of the urinary mercury concentration for creatinine concentration, and stability of exposure conditions (examined subjects were exposed to mercury vapor for at least 1 year). Strong correlations were found between the daily intensity of exposure to mercury vapor and the end of workshift levels in blood ($r=0.86$; $n=40$) or urine ($r=0.81$; $n=34$). These relationships indicated a conversion factor of 1:4.5 (air:blood) and 1:1.22 (air:urine as ug/g creatinine). These factors were used

to extrapolate blood or urine levels associated with effects in the reported studies to airborne mercury levels.

Sensory and motor nerve conduction velocities were studied in 18 workers from a mercury cell chlorine plant (Levine et al., 1982). Time-integrated urine Hg levels were used as an indicator of mercury exposure. Using linearized regression analysis, the authors found that motor and sensory nerve conduction velocity changes (i.e., prolonged distal latencies correlated with the time-integrated urinary Hg levels in asymptomatic exposed workers) occurred when urinary Hg levels exceeded 25 ug/L. This study demonstrates that mercury exposure can be associated with preclinical evidence of peripheral neurotoxicity.

Singer et al. (1987) studied nerve conduction velocity of the median motor, median sensor and sural nerves in 16 workers exposed to various inorganic mercury compounds (e.g., mercuric oxides, mercurial chlorides, and phenyl mercuric acid) for an average of 7.3 +/- 7.1 years as compared with an unexposed control group using t-tests. They found a slowing of nerve conduction velocity in motor, but not sensory, nerves that correlated with increased blood and urine Hg levels and an increased number of neurologic symptoms. The mean mercury levels in the exposed workers were 1.4 and 10 ug/L for blood and urine, respectively. These urine levels are 2-fold less than those associated with peripheral neurotoxicity in other studies (e.g., Levine et al., 1982). There was considerable variability in the data presented by Singer et al. (1987), however, and the statistical analyses (t-test) were not as rigorous as those employed by Levine et al. (1982) (linearized regression analysis). Furthermore, the subjects in the Levine et al. (1982) study were asymptomatic at higher urinary levels than those reported to be associated with subjective neurological complaints in the workers studied by Singer et al. (1987). Therefore, these results are not considered to be as reliable as those reported by Levine et al. (1982).

Miller et al. (1975) investigated several subclinical parameters of neurological dysfunction in 142 workers exposed to inorganic mercury in either the chloralkali industry or a factory for the manufacture of magneti materials. They reported a significant increase in average forearm tremor frequency in workers whose urinary Hg concentrations exceeded 50 ug/L as compared with unexposed controls. Also observed were eyelid fasciculation, hyperactive deep-tendon reflexes and dermatographia, but there was no correlation between the incidence of these findings and urinary Hg levels.

Roels et al. (1985) examined 131 male and 54 female workers occupationally exposed to mercury vapor for an average duration of 4.8 years. Urinary mercury (52 and 37 ug/g creatinine for males and females, respectively) and blood mercury levels (14 and 9 ug/L for males and females, respectively) were recorded, but atmospheric mercury concentration was not provided. Symptoms indicative of CNS disorders were reported but not related to mercury exposure. Minor renal tubular effects were detected in mercury-exposed males and females and attributed to current exposure intensity rather (urinary Hg >50 ug/g creatinine) than exposure duration. Male subjects with urinary mercury levels of >50 ug/g creatinine exhibited preclinical signs of hand tremor. It was noted that females did not exhibit this effect and that their urinary mercury never reached the level of 50 ug/g creatinine. A companion study (Roels et al., 1987) related air mercury (Hg-air)levels to blood mercury (Hg-blood) and urinary mercury (Hg-U) values in 10 workers in a chloralkali battery plant. Duration of exposure was not specified. A high correlation was reported for Hg-air and Hg-U for preshift exposure ($r=0.70$, $p<0.001$) and post-shift ($r=0.81$, $p<0.001$) measurements. Based on these data and the results of their earlier (1985) study, the investigators suggested that some mercury-induced effects may occur when Hg-U levels exceed 50 ug/g creatinine, and that this value corresponds to a mercury TWA of about 40 ug/cu.m.

A survey of 567 workers at 21 chloralkali plants was conducted to ascertain the effects of mercury vapor inhalation (Smith et al., 1970). Mercury levels ranged from <0.01 to 0.27 mg/cu.m and chlorine concentrations ranged from 0.1 to 0.3 ppm at most of the working stations of these plants. Worker exposure to mercury levels (TWA) varied, with 10.2% of the workers being exposed to <0.01 mg/cu.m, 48.7% exposed to 0.01 to 0.05 mg/cu.m, 25.6% exposed to 0.06 to 0.10 mg/cu.m and 4.8% exposed to 0.24 to 0.27 mg/cu.m (approximately 85% were exposed to Hg levels less than or equal to 0.1 mg/cu.m). The duration of employment for the examined workers ranged from one year (13.3%) to >10 years (31%), with 55.7% of the workers being employed for 2 or 9 years. A group of 600 workers not exposed to chlorine served as a control group for assessment of chlorine effects, and a group of 382 workers not exposed to either chlorine or mercury vapor served as the reference control group. A strong positive correlation ($p<0.001$) was found between the mercury TWAs and the reporting of subjective neuropsychiatric symptoms (nervousness, insomnia), occurrence of objective tremors, and weight and appetite loss.

A positive correlation ($p<0.001$) was also found between mercury exposure levels and urinary and blood mercury levels of test subjects. No adverse alterations in cardiorespiratory, gastrointestinal, renal or hepatic functions were attributed to the mercury vapor exposure. Additionally, biochemical (hematologic data, enzyme activities) and clinical measurements (EKG, chest X-rays) were no different between the mercury-exposed and non-exposed workers. No significant signs or symptoms were noted for individuals exposed to mercury vapor concentrations less than or equal to 0.1 mg/cu.m. This study provides data indicative of a NOAEL of 0.1 mg Hg/cu.m and a LOAEL of 0.18 mg Hg/cu.m. In a followup study conducted by Bunn et al. (1986), however, no significant differences in the frequency of objective or subjective findings such as weight loss and appetite loss were observed in workers exposed to mercury at levels that ranged between 50 and 100 ug/L. The study by Bunn et al. (1986) was limited, however, by the lack of information provided regarding several methodological questions such as quality assurance measures and control of possible confounding variables.

The mercury levels reported to be associated with preclinical and symptomatic neurological dysfunction are generally lower than those found to affect kidney function, as discussed below.

Piikivi and Ruokonen (1989) found no evidence of glomerular or tubular damage in 60 chloralkali workers exposed to mercury vapor for an average of 13.7 +/- 5.5 years as compared with their matched referent controls. Renal function was assessed by measuring urinary albumin and N-acetyl-beta-glucosaminidase (NAG) activity. The mean blood Hg level in the exposed workers was 14 ug/L and the mean urinary level was 17 ug/L. The authors extrapolated the NOAEL for kidney effects based on these results of 0.025 mg/cu.m from blood levels using the conversion factor calculated by Roels et al. (1987).

Stewart et al. (1977) studied urinary protein excretion in 21 laboratory workers exposed to 10-50 ug/cu.m of mercury. Their urinary level of mercury was about 35 ug/L. Increased proteinuria was found in the exposed workers as compared with unexposed controls. When preventive measure were instituted to limit exposure to mercury, proteinuria was no longer observed in the exposed technicians.

Lauwerys et al. (1983) found no change in several indices of renal function (e.g., proteinuria, albuminuria, urinary excretion of retinol-binding protein, aminoaciduria, creatinine in serum, beta-2-microglobulin in serum) in 62 workers exposed to mercury vapor for an average of 5.5 years. The mean urinary Hg excretion in the exposed workers was 56 ug/g creatinine, which corresponds to an exposure level of about 46 ug/cu.m according to a conversion factor of 1:1.22 (air:urine [ug/g creatinine]) (Roels et al., 1987). Despite the lack of observed renal effects, 8 workers were found to have an increased in serum anti-laminin antibodies, which can be indicative of immunological effects. In a followup study conducted by Bernard et al. (1987), however, there was no evidence of increased serum anti-laminin antibodies in 58 workers exposed to mercury vapor for an average of 7.9 years. These workers had a mean urinary Hg excretion of 72 ug/g creatinine, which corresponds to an exposure levels of about 0.059 mg/cu.m.

Stonard et al. (1983) studied renal function in 100 chloralkali workers exposed to inorganic mercury vapor for an average of 8 years. No changes in the following urinary parameters of renal function were observed at mean urinary Hg excretion rates of 67 ug/g creatinine: total protein, albumin, alpha-1-acid glycoprotein, beta-2-microglobulin, NAG, and gamma-glutamyl transferase. When urinary Hg excretion exceeded 100 ug/g creatinine, a small increase in the prevalence of higher activities of NAG and gamma-glutamyl transferase was observed.

The mercury levels reported to be associated with preclinical and symptomatic neurological dysfunction and kidney effects are lower than those found to pulmonary function, as discussed below.

McFarland and Reigel (1978) described the cases of 6 workers who were acutely exposed (4-8 hours) to calculated metallic mercury vapor levels of 1.1 to 44 mg/cu.m. These men exhibited a combination of chest pains, dyspnea, cough, hemoptysis, impairment of pulmonary function (reduced vital capacity), diffuse pulmonary infiltrates and evidence of interstitial pneumonitis. Although the respiratory symptoms resolved, all six cases exhibited chronic neurological dysfunction, presumably as a result of the acute, high-level exposure to mercury vapor.

Lilis et al. (1985) described the case of a 31-year-old male who was acutely exposed to high levels of mercury

vapor in a gold-extracting facility. Upon admission to the hospital, the patient exhibited dyspnea, chest pain with deep inspiration, irregular infiltrates in the lungs and reduced pulmonary function (forced vital capacity [FVC]). The level of mercury to which he was exposed is not known, but a 24-hour urine collection contained 1900 ug Hg/L. Although the patient improved gradually over the next several days, 11 months after exposure he still showed signs of pulmonary function abnormalities (e.g., restriction and diffusion impairment).

Levin et al. (1988) described four cases of acute high-level mercury exposure during gold ore purification. The respiratory symptoms observed in these four cases ranged from minimal shortness of breath and cough to severe hypoxemia. The most severely affected patient exhibited mild interstitial lung disease both radiographically and on pulmonary function testing. One patient had a urinary Hg level of 245 ug/L upon hospital admission. The occurrence of long-term respiratory effects in these patients could not be evaluated since all but one refused follow-up treatment.

Ashe et al. (1953) reported that there was no histopathological evidence of respiratory damage in 24 rats exposed to 0.1 mg Hg/cu.m 7 hr/day, 5 days/week for 72 weeks. This is equivalent to a NOAEL[HEC] of 0.07 mg/cu.m.

Kishi et al. (1978) observed no histopathological changes in the lungs of rats exposed to 3 mg/cu.m of mercury vapor 3 hours/day, 5 days/week for 12-42 weeks.

Beliles et al. (1967) observed no histopathological changes in the lungs of pigeons exposed to 0.1 mg/cu.m of mercury vapor 6 hours/day, 5 days/week for 20 weeks.

Neurological signs and symptoms (i.e., tremors) were observed in 79 workers exposed to metallic mercury vapor whose urinary mercury levels exceeded 500 ug/L. Short-term memory deficits were reported in workers whose urine levels were less than 500 ug/L (Langolf et al., 1978).

Impaired performance in mechanical and visual memory tasks and psychomotor ability tests was reported by Forzi et al. (1978) in exposed workers whose urinary Hg levels exceeded 100 ug/L.

Decreased strength, decreased coordination, increased tremor, decreased sensation and increased prevalence of Babinski and snout reflexes were exhibited by 247 exposed workers whose urinary Hg levels exceeded 600 ug/L. Evidence of clinical neuropathy was observed at urinary Hg levels that exceeded 850 ug/L (Albers et al., 1988).

Preclinical psychomotor dysfunction was reported to occur at a higher incidence in 43 exposed workers (mean exposure duration of 5 years) whose mean urinary excretion of Hg was 50 ug/L. Workers in the same study whose mean urinary Hg excretion was 71 ug/L had a higher incidence of total proteinuria and albuminuria (Roels et al., 1982).

Postural and intention tremor was observed in 54 exposed workers (mean exposure duration of 7.7 years) whose mean urinary excretion of Hg was 63 ug/L (Roels et al., 1989).

Verbeck et al. (1986) observed an increase in tremor parameters with increasing urinary excretion of mercury in 21 workers exposed to mercury vapor for 0.5-19 years. The LOAEL for this effect was a mean urinary excretion of 35 ug/g creatinine.

Rosenman et al. (1986) evaluated routine clinical parameters (physical exams, blood chemistry, urinalysis), neuropsychological disorders, urinary NAG, motor nerve conduction velocities and occurrence of lenticular opacities in 42 workers of a chemical plant producing mercury compounds. A positive correlation ($p<0.05$ to $p<0.001$) was noted between urinary mercury (levels ranged from 100-250 ug/L) and the number of neuropsychological symptoms, and NAG excretions and the decrease in motor nerve conduction velocities.

Evidence of renal dysfunction (e.g., increased plasma and urinary concentrations of beta-galactosidase, increased urinary excretion of high-molecular weight proteins and a slightly increased plasma beta-2-microglobulin concentration) was observed in 63 chloralkali workers. The incidence of these effects increased in workers whose urinary Hg excretion exceeded 50 ug/g creatinine (Buchet et al., 1980).

Increased urinary NAG levels were found in workers whose urinary Hg levels exceeded 50 ug/L (Langworth et al., 1992).

An increase in the concentration of urinary brush border proteins (BB-50) was observed in 20 workers whose mean urinary Hg excretion exceeded 50 ug/g creatinine (Mutti et al., 1985).

Foa et al. (1976) found that 15 out of 81 chloralkali workers exposed to 60-300 ug/cu.m mercury exhibited proteinuria.

An increased excretion of beta-glutamyl transpeptidase, indicative of renal dysfunction, was found in 509 infants dermally exposed to phenylmercury via contaminated diapers (Gotelli et al., 1985).

Berlin et al. (1969) exposed rats, rabbits and monkeys to 1 mg/cu.m of mercury vapor for 4 hours and measured the uptake and distribution of mercury in the brain as compared with animals injected intravenously with the same doses of mercury as mercuric salts. Mercury accumulated in the brain following inhalation exposure to metallic mercury vapor at levels that were 10 times higher than those observed following intravenous injection of the same dose of mercury as mercuric salts. These results demonstrate that mercury is taken up by the brain following inhalation of the vapor at higher levels than other forms of mercury and that this occurs in all species studied.

Limited animal studies concerning inhalation exposure to inorganic mercury are available. The results of a study conducted by Baranski and Szymczyk (1973) were reported in an English abstract. Adult female rats were exposed to metallic mercury vapor at 2.5 mg/cu.m for 3 weeks prior to fertilization and during gestation days 7-20. A decrease in the number of living fetuses was observed in the dams compared with unexposed controls, and all pups born to the exposed dams died by the sixth day after birth. However, no difference in the occurrence of developmental abnormalities was observed between exposed and control groups. The cause of death of the pups in the mercury-exposed group was unknown, although an unspecified percentage of the deaths was attributed by the authors to a failure of lactation in the dams. Death of pups was also observed in another experiment where dams were only exposed prior to fertilization (to 2.5 mg/cu.m), which supports the conclusion that the high mortality in the first experiment was due at least in part to poor health of the mothers. Without further information, this study must be considered inconclusive regarding developmental effects.

The only other study addressing the developmental toxicology of mercury is the one reported in abstract form by Steffek et al. (1987) and, as such, is included as a supporting study. Sprague-Dawley rats (number not specified) were exposed by inhalation to mercury vapor at concentrations of 0.1, 0.5 or 1.0 mg/cu.m throughout the period of gestation (days 1-20) or during the period of organogenesis (days 10-15). The authors indicated the exposure protocols to be chronic and acute exposure, respectively. At either exposure protocol, the lowest mercury level produced no detectable adverse effect. At 0.5 mg/cu.m, an increase in the number of resorptions (5/41) was noted for the acute group, and two of 115 fetuses exhibited gross cranial defects in the chronic group. At 1.0 mg/cu.m, the number of resorptions was increased in acute (7/71) and chronic (19/38) groups and a decrease in maternal and fetal weights also was detected in the chronic exposure group. No statistical analysis for these data was provided. A LOAEL of 0.5 mg/cu.m is provided based on these data.

Mishinova et al. (1980) investigated the course of pregnancy and parturition in 349 women exposed via inhalation to metallic mercury vapors (unspecified concentrations) in the workplace as compared to 215 unexposed women. The authors concluded that the rates of pregnancy and labor complication were high among women exposed to mercury and that the effects depended on "the length of service and concentration of mercury vapors." Lack of sufficient details preclude the evaluation of dose-response relationships.

In a questionnaire that assessed the fertility of male workers exposed to mercury vapor, Lauwerys et al. (1985) found no statistically significant change in the observed number of children born to the exposed group compared with a matched control group. The urinary excretion of mercury in the exposed workers ranged from 5.1 to 272.1 ug/g creatinine.

Another study found that exposure to metallic mercury vapor caused prolongation of estrus cycles in animals. Baranski and Szymczyk (1973) reported that female rats exposed via inhalation to mercury vapor at an average of

2.5 mg/cu.m, 6 hours/day, 5 days/week for 21 days experienced longer estrus cycles than unexposed animals. In addition, estrus cycles during mercury exposure were longer than normal estrus cycles in the same animals prior to exposure. Although the initial phase of the cycle was protracted, complete inhibition of the cycle did not occur. During the second and third weeks of exposure, these rats developed signs of mercury poisoning including restlessness, seizures and trembling of the entire body. The authors speculated that the effects on the estrus cycle were caused by the action of mercury on the CNS (i.e., damage to the hypothalamic regions involved in the control of estrus cycling).

Renal toxicity has been reported following oral exposure to inorganic mercury salts in animals, with the Brown-Norway rat appearing to be uniquely sensitive to this effect. These mercury-induced renal effects in the Brown-Norway rat are the basis for the oral RfD for mercurial mercury. Several investigators have produced autoimmune glomerulonephritis by administering HgCl2 to Brown-Norway rats (Druet et al., 1978).

The current OSHA standard for mercury vapor is 0.05 mg/cu.m. NIOSH recommends a TWA Threshold Limit Value of 0.05 mg/cu.m for mercury vapor.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study -- Medium
Data Base -- Medium
RfC -- Medium

Due to the use of a sufficient number of human subjects, the inclusion f appropriate control groups, the exposure duration, the significance level of the reported results and the fact that exposure levels in a number of the studies had to be extrapolated from blood mercury levels, confidence in the key studies is medium. The LOAEL values derived from these studies can be corroborated by other human epidemiologic studies. The adverse effects reported in these studies are in accord with the well-documented effects of mercury poisoning. The lack of human or multispecies reproductive/ developmental studies precludes assigning a high confidence rating to the data base and inadequate quantification of exposure levels. Based on these considerations, the RfC for mercury is assigned a confidence rating of medium.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- U.S. EPA, 1995

This IRIS summary is included in The Mercury Study Report to Congress which was reviewed by OHEA and EPA's Mercury Work Group in November 1994. An interagency review by scientists from other federal agencies took place in January 1995. The report was also reviewed by a panel of non-federal external scientists in January 1995 who met in a public meeting on January 25-26. All reviewers comments have been carefully evaluated and considered in the revision and finalization of this IRIS summary. A record of these comments is summarized in the IRIS documentation files.

Other EPA Documentation -- None

Agency Work Group Review -- 11/16/1989, 03/22/1990, 04/19/1990

Verification Date -- 04/19/1990

___I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 05/01/1995

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Preparation Date -- 5/24/94

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on inadequate human and animal data. Epidemiologic studies failed to show a correlation between exposure to elemental mercury vapor and carcinogenicity; the findings in these studies were confounded by possible or known concurrent exposures to other chemicals, including human carcinogens, as well as lifestyle factors (e.g., smoking). Findings from genotoxicity tests are severely limited and provide equivocal evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. A number of epidemiological studies were conducted that examined mortality among elemental mercury vapor-exposed workers. Conflicting data regarding a correlation between mercury exposure and an increased incidence of cancer mortalities have been obtained. All of the studies have limitations that complicate interpretation of their results for associations between mercury exposure and induction of cancer; increased cancer rates were attributable to other concurrent exposures or lifestyle factors.

A retrospective cohort study examined mortality among 5663 white males who worked between 1953 and 1963 at a plant in Oak Ridge, Tennessee, where elemental mercury was used for lithium isotope separation (Cragle et al., 1984). The workers were divided into three cohorts: exposed workers who had been monitored on a quarterly basis for mercury levels in urine (n=2,133); workers exposed in the mercury process section for whom urinalysis monitoring data were not collected (n=270); and unexposed workers from other sections of the nuclear weapons production facility (n=3260). The study subjects worked at least 4 months during 1953-1958 (a period when mercury exposures were likely to be high); mortality data from death certificates were followed through the end of 1978. The mean age of the men at first employment at the facility was 33 years, and the average length of their employment was >16 years with a mean of 3.73 years of estimated mercury exposure. Air mercury levels were monitored beginning in 1955; during 1955 through the third quarter of 1956, air mercury levels were reportedly

above 100 ug/cu.m in 30-80% of the samples. Thereafter, air mercury levels decreased to concentrations below 100 ug/cu.m. The mortality experience (i.e., the SMR) of each group was compared with the age-adjusted mortality experience of the U.S. white male population. Among exposed and monitored workers, no significant increases in mortality from cancer at any site were reported, even after the level or length of exposure was considered. A significantly lower mortality from all causes was observed. An excessive number of deaths was reportedly due to lung cancer in the exposed and monitored workers (42 observed, 31.36 expected), but also in the unexposed workers (71 observed, 52.93 expected). The SMR for each group was 1.34; the elevated incidence of lung cancer deaths was, therefore, attributed to some other factor at the plant and/or to lifestyle factors (e.g., smoking) common to both the exposed and unexposed groups. Study limitations include small cohort sizes for cancer mortality, which limited the statistical stability of many comparisons.

Barregard et al. (1990) studied mortality and cancer morbidity between 1958 and 1984 in 1190 workers from eight Swedish chloralkali plants that used the mercury cell process in the production of chlorine. The men included in the study had been monitored for urinary or blood mercury for more than one year between 1946 and 1984. Vital status and cause of death were ascertained from the National Population Register and the National Bureau of Statistics. The cancer incidence of the cohort was obtained from the Swedish Cancer Register. The observed total mortality and cancer incidences were compared with those of the general Swedish male population. Comparisons were not made between exposed and unexposed workers. Mean urinary mercury levels indicated a decrease in exposure between the 1950s and 1970s; the mean urinary mercury level was 200 ug/L during the 1950s, 150 ug/L during the 1960s and 50 ug/L in the 1970s. Mortality from all causes was not significantly increased in exposed workers. A significant increase in deaths from lung tumors was observed in exposed workers 10 years or more after first exposure (rate ratio, 2.0; 95% CI, 1.0-3.8). Nine of the 10 observed cases of lung cancer occurred among workers (457 of the 1190) possibly exposed to asbestos as well as to mercury. No dose response was observed with respect to mercury exposure and lung tumors. This study is limited because no quantitation was provided on smoking status, and results were confounded by exposure to asbestos.

Ahlbom et al. (1986) examined the cancer mortality during 1961-1979 of cohorts of Swedish dentists and dental nurses aged 20-64 and employed in 1960 (3454 male dentists, 1125 female dentists, 4662 female dental nurses). Observed incidences were compared with those expected based on cancer incidence during 1961-1979 among all Swedes employed during 1960 and the proportion of all Swedes employed as dentists and dental nurses. Data were stratified by sex, age (5-year age groups) and county. The incidence of glioblastomas among the dentists and dental nurses combined was significantly increased compared to survival rates (SMR, 2.1; 95% CI, 1.3-3.4); the individual groups had apparently elevated SMRs (2.0-2.5), but the 95% confidence intervals of these groups included unity. By contrast, physicians and nurses had SMRs of only 1.3 and 1.2, respectively. Exposure to mercury could not be established as the causative factor because exposure to other chemicals and X-rays was not ruled out.

Amandus and Costello (1991) examined the association between silicosis and lung cancer mortality between 1959 and 1975 in 9912 white male metal miners employed in the United States between 1959 and 1961. Mercury exposures were not monitored. Exposures to specific metals among the silicotic and nonsilicotic groups were analyzed separately. Lung cancer mortality in both silicotic and nonsilicotic groups was compared with rates in white males in the U.S. population. Both silicotic (n=11) and nonsilicotic mercury miners (n=263) had significantly increased lung cancer mortality (SMR, 14.03; 95% CI, 2.89-40.99 for silicotics. SMR, 2.66; 95% CI, 1.15-5.24 for nonsilicotics). The analysis did not focus on mercury miners, and confounders such as smoking and radon exposure were not analyzed with respect to mercury exposure. This study is also limited by the small sample size for non-silicotic mercury miners.

A case-control study of persons admitted to a hospital in Florence, Italy, with lung cancer between 1981-1983 was performed to evaluate occupational risk factors (Buiatti et al., 1985). Cases were matched with one or two controls (persons admitted to the hospital with diagnoses other than lung cancer or suicide) with respect to sex, age, date of admission and smoking status. Women who had "ever worked" as hat makers had a significantly increased risk of lung cancer. The duration of employment as a hat maker averaged 22.2 years, and latency averaged 47.8 years. Workers in the Italian hat industry were known to be occupationally exposed to mercury; however, the design of this study did not allow evaluation of the relationship between cumulative exposure and cancer incidence. In addition, interpretation of the results of this study is limited by the small sample size (only 6/376 cases reported this occupation) and by exposure of hat makers to other pollutants including arsenic, a known lung carcinogen.

Ellingsen et al. (1992) examined the total mortality and cancer incidence among 799 workers employed for more than 1 year in two Norwegian chloralkali plants. Mortality incidence between 1953 and 1988 and cancer incidence between 1953 and 1989 were examined. Mortality and cancer incidence were compared with that of the age-adjusted general male Norwegian population. No increase in total cancer incidence was reported, but lung cancer was significantly elevated in the workers (rate ratio, 1.66; 95% CI, 1.0-2.6). No causal relationship can be drawn from the study between mercury exposure and lung cancer because no correlation existed between cumulative mercury dose, years of employment or latency time. Also, the prevalence of smoking was 10 20% higher in the exposed workers, and many workers were also exposed to asbestos.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Druckrey et al. (1957) administered 0.1 mL of metallic mercury to 39 male and female rats (BD III and BD IV strains) via intraperitoneal injection. Among the rats surviving longer than 22 months, 5/12 developed peritoneal sarcomas. The increase in the incidence of sarcomas was observed only in those tissues that had been in direct contact with the mercury. Although severe kidney damage was reported in all treated animals, no renal tumors or tumors at any site other than the peritoneal cavity were observed.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Cytogenetic monitoring studies of workers occupationally exposed to mercury by inhalation provide very limited evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells. Popescu et al. (1979) compared four men exposed to elemental mercury vapor with an unexposed group and found a statistically significant increase in the incidence of chromosome aberrations in the WBCs from whole blood. Verschaeve et al. (1976) found an increase in aneuploidy after exposure to low concentrations of vapor, but results could not be repeated in later studies (Verschaeve et al., 1979). Mabille et al. (1984) did not find increases in structural chromosomal aberrations of lymphocytes of exposed workers. Similarly, Barregard et al. (1991) found no increase in the incidence or size of micronuclei and no correlation between micronuclei and blood or urinary mercury levels of chloralkali workers. A statistically significant correlation was observed between cumulative exposure to mercury and micronuclei induction in T lymphocytes in exposed workers, suggesting a genotoxic effect.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

None.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

None.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source document -- U.S. EPA, 1995

This IRIS summary is included in The Mercury Study Report to Congress which was reviewed by OHEA and EPA's Mercury Work Group in November 1994. An interagency review by scientists from other federal agencies took place in January 1995. The report was also reviewed by a panel of non-federal external scientists in January 1995 who met in a public meeting on January 25-26. All reviewers comments have been carefully evaluated and considered in the revision and finalization of this IRIS summary. A record of these comments is summarized in the IRIS documentation files.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 01/13/1988, 03/03/1994

Verification Date -- 03/03/1994

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 06/01/1995

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None

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VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6

Date	Section	Description
09/07/1988	II.	Carcinogen summary on-line
09/01/1989	VI.	Bibliography on-line
12/01/1989	I.B.	Inhalation RfD now under review
05/01/1991	II.A.3.	Text edited
01/01/1992	IV.	Regulatory Action section on-line
04/01/1994	II.	Carcinogenicity assessment noted as pending change
04/01/1994	II.D.2.	Work group review date added
05/01/1995	All	Name changed from mercury (inorganic)
05/01/1995	II.	Carcinogen assessment replaced
05/01/1995	VI.C.	Carcinogen assessment references replaced
06/01/1995	I.B.	Inhalation RfC summary on-line
06/01/1995	VI.B.	Inhalation RfC references on-line

VIII. SYNONYMS

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 05/01/1995

7439-97-6
hydragyrum
Mercury
Mercury, elemental
Mercury, inorganic
Mercury, metallic
Mercury (organo) alkyl compounds
Caswell No. 546
COLLOIDAL MERCURY
EPA Pesticide Chemical Code 052301

KWIK [Dutch]
Liquid Silver
Mercure [French]
Mercurio [Italian]
Mercurio [Spanish]
Mercury compounds
Mercury vapor
NCI-C60399
Quecksilber [German]
Quicksilver

Dimethyl phthalate; CASRN 131-11-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Dimethyl phthalate

File On-Line 09/07/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	03/01/1994
Inhalation RfC Assessment (I.B.)	message	10/01/1990
Carcinogenicity Assessment (II.)	on-line	02/01/1993

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Dimethyl phthalate
CASRN -- 131-11-3

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Dimethyl phthalate
CASRN -- 131-11-3

The health effects data for dimethylphthalate were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for derivation of an inhalation RfC. The verification status of this chemical is currently not verifiable. For additional information on health effects of this chemical, interested parties are referred to the EPA documentation listed below.

U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters Prepared by the Office of Health and Environmental Assessement, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)

Agency Work Group Review -- 07/26/1990

EPA Contacts:

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Dimethyl phthalate
CASRN -- 131-11-3
Last Revised -- 02/01/1993

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable.

Basis -- Pertinent data regarding carcinogenicity was not located in the available literature.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. A 2-year dietary study in rats by Lehman (1955) was not designed to measure carcinogenic effects.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

DMP was found to be a weak direct-acting mutagen in forward and reverse mutation assays in Salmonella typhimurium (Seed, 1982; Rubin et al., 1979, Kozumbo et al., 1982). DMP was active in the mouse lymphoma forward mutation assay only in the presence of metabolic activation (CMA, 1986). Negative results were found in a mouse dominant lethal test (Yurchenko and Gleiberman, 1980).

In vitro assays showed that liver homogenate-associated esterases hydroloyzed DMP to methanol and to the monoester which has been shown to be a nonmutagenic compound in Salmonella assay and to methanol (Kozumbo et al., 1982). Other research also indicates that DMP is hydrolyzed to monoesters (Kaneshima et al., 1978; Rowland, 1977; Albro and Moore, 1974).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1980, 1987

The 1987 Drinking Water Criteria Document for Phthalic Acid Esters has received OHEA review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 08/26/1987

Verification Date -- 08/26/1987

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Dimethyl phthalate
CASRN -- 131-11-3
Last Revised -- 10/01/1990

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfD REFERENCES

U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters Prepared by the Office of Health and Environmental Assessement, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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U.S. EPA. 1987. *Drinking Water Criteria Document for Phthalic Acid Esters.* Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. External Review Draft.

_VII. REVISION HISTORY

Substance Name -- Dimethyl phthalate
CASRN -- 131-11-3

Date	Section	Description
09/07/1988	II.	Carcinogen summary on-line
04/01/1989	V.	Supplementary data on-line
03/01/1990	VI.	Bibliography on-line
09/01/1990	I.B.	Not verified; data inadequate
10/01/1990	I.B.	Inhalation RfC text modified
10/01/1990	VI.B.	Bibliography on-line
08/01/1991	II.D.3.	Primary and secondary contacts changed
01/01/1992	IV.	Regulatory Action section on-line
02/01/1993	II.D.3.	Primary contact changed
03/01/1994	I.A.	Work group review date added

VIII. SYNONYMS

Substance Name -- Dimethyl phthalate
CASRN -- 131-11-3
Last Revised -- 09/07/1988

131-11-3
1,2-benzenedicarboxylic acid, dimethyl ester
dimethyl 1,2-benzenedicarboxylate
dimethyl benzene-o-dicarboxylate
Dimethyl phthalate
DMP
methyl phthalate
phthalic acid, dimethyl ester